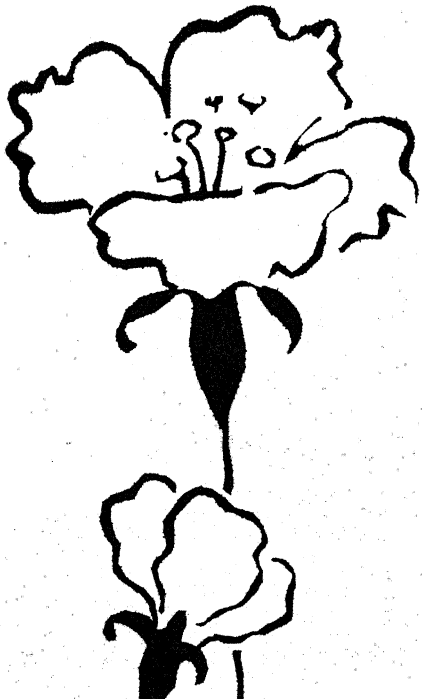
"ROLE OF ADMISSION TEST AS A SCREENING PROCEDURE FOR PERINATAL OUTCOME"

THESIS
FOR

DOCTOR OF MEDICINE
(OBSTETRICS & GYNAECOLOGY)

BUNDELKHAND UNIVERSITY,
JHANSI (U.P.)

2005 DR. POOJA GUPTA



Dedicated to My Father

CERTIFICATE

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She has put in the necessary stay in the department as per required by the regulation of Bundelkhand University

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Finally I thank my patients whose kind cooperation made this study possible.

Last, but certainly not the least, I pay my sincere prayer to the ALMIGHTY GOD who gave me power, blessings and encouragement to accomplish this task.

Jhansi
Dated : SEPT '04

Pooja

(Dr. Pooja Gupta)

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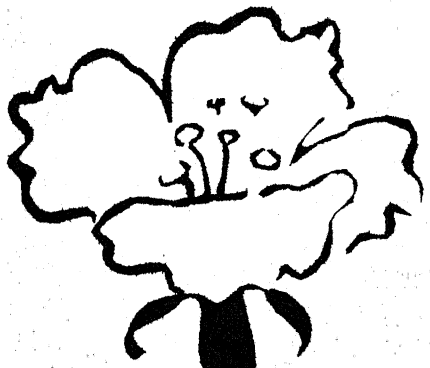

Introduction

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Childhood neurological handicap has commonly been ascribed to various intrapartum events leading to birth asphyxia. A few neurological deficits due to congenital malformations, genetically inherited disorders or chromosomal aberrations are inevitable but majority of them are avoidable and therefore the need of strict and deligent antepartum and intrapartum fetal monitoring.

Fetal monitoring can be done by simple methods such as daily fetal movement count and intermittent auscultation as well as by sophisticated electronic gadgets. Fetal movement count has been an important means to assess fetal well being in earlier days, but later it was recognized that mothers perceived only 16% of fetal movements recorded by Doppler device and fetal movements lasting more 20 sec. were identified more accurately by the mother than shorter episodes of fetal activity.

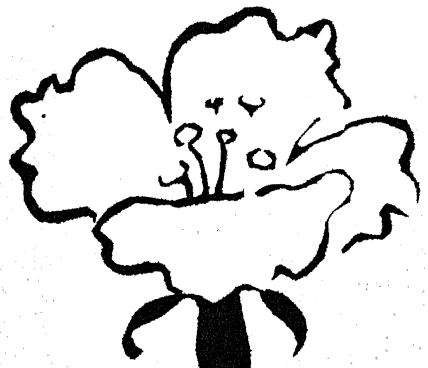
Intermittent auscultation provides an acceptable means of monitoring during labour. But it needs intensive supervision and only baseline fetal heart rate can be measured and other features like baseline variability, acceleration & deceleration are difficult to quantify.

Routine electronic monitoring of fetal heart rate in labour is becoming an established practice in modern obstetrics, though with a few disadvantage. Some mothers desiring natural childbirth are reluctant to accept continuous monitoring. Also economic constraints in most parts of the world limit the above. It may also increase the rate of cesarean section according to a few studies.

Selection of patients for continuous monitoring or intermittent auscultation is therefore necessary for which an antenatal risk classification is used in most of the set ups. Unfortunately risk assessment profiles are often an insufficient tool for selection. Intrapartum fetal morbidity and mortality are not uncommon in a low risk population and FHR changes and fetal acidosis might occur with the same frequency as in a high risk group.

Admission test, a non stress test done at admission in prelabour or early labour is one such alternative to labeling patients for FHR monitoring on the basis of an antenatal risk classification. Based on the assumption that early uterine contractions may serve as a functional stress to the fetus, an admission test might detect fetal intrauterine asphyxia present already at admission and might have some predictive value of asphyxia that may develop in labor. It is a simple, non-invasive and short fetal heart rate recording at the time of admission. It thus avoids unnecessary delay in intervention in an already compromised fetus at the time of admission.

With the above aim, this study has been planned to evaluate the value of admission test in predicting complication or distress going to be developed during labor and correlating the finding of admission test with fetal outcome parameters.



Review of Literature

Review Of Literature

INTRAUTERINE ASSESSMENT OF FETAL WELL BEING

Since intrapartum events are estimated for 20% of still births, 20-40% of cerebral palsy and 10% of severe mental retardation and it has been observed that intrapartum fetal hypoxia is one of the potential factors involved in the developments of handicaps and perinatal deaths. Goal has been to identify fetus at risk by antepartum and intrapartum fetal monitoring.

No written records of detection of fetal life in utero exist in western literature until seventeenth century. It was not until 1818 that Francis Isaac Mayor of Geneva, a surgeon reported that fetal heart can be audible different from maternal pulse by applying the ear directly to the pregnant mother's abdomen. Ferguson was the first person in British Isles to describe fetal heart sounds in 1827. He influenced Evory Kennedy, assistant master at Rotunda lying in hospital in Dublin, who wrote famous work entitled – "observation on obsteric auscultation" in 1833.

There was much argument over the technique of listening. Anton Friedrich Hohl was first to describe the design of fetal stethoscope in 1834. Depaul modifies this describing both in his – Traite theorique et pratigue d'ascultation Obstetricale in 1847. Although Penard's Name is most commonly associated with stethoscope, his version followed much later, appearing in 1876. In 1849 Kilan proposed. The stethoscopical indications of forceps operation". Wrinkel in 1893 empirically set the limits of normal heart rate at 120 bpm to 160 bpm.

II. SIGNIFICANCE OF FETAL MOVEMENT COUNT

Passive unstimulated activity of human fetus commences as early as 7 weeks of gestation and becomes more sophisticated and coordinated by the

end of pregnancy (Vindla and James, 1995). Indeed, beyond eight menstrual weeks, fetal body movements are never absent for time periods exceeding 13 minutes (De Vries and coworkers, 1985). Between 20 and 30 weeks, general body movements become organized and fetus starts to show rest activity cycles (Soronkin and coworkers, 1982). In the third trimester, fetal movement maturation continues until about 36 weeks, when behavioural states are established in 80% of normal fetuses. Nijhuis and colleagues (1982) studied fetal heart rate patterns, general body movements, and eye movements and described four fetal behavioural states.

State 1F: is a quiescent state (quite sleep), with narrow, oscillatory bandwidth of the fetal heart rate.

State 2F: includes frequent gross body movements, continuous eye movement and wider oscillation of fetal heart rate. This state is analogous to REM (rapid eye movement) or active sleep in the neonate.

State 3F: includes continuous eye movements in the absence of body movements and no accelerations of heart rate. The existence of this state is disputed (Pillai and James, 1990a).

State 4F: is one of vigorous body movment with continuous eye movements and fetal heart rate accelerations. This state corresponds to the awake state in infants.

Fetuses spend most of their time in states 1F and 2F for example, at 38 weeks gestational age, fetuses spend 75% of their time in states 1F and 2F (Nejuis and Colleagues, 1982).

An important determinant of fetal activity appears to be sleep – awake cycles which are independent of the maternal sleep- awake state. "Sleep

Cyclicity" has been described to vary from about 20 minutes to as much as 75 minutes.

Sadovsky and colleagues (1979b) studied the type of fetal movements in 120 normal pregnancies and classified the movements into three categories according to both maternal perception and independent recording using piezoelectric sensors. Weak, strong and rolling movements throughout the last half of pregnancy were quantified.

The mean number of weekly movements calculated from 12-hour daily recording periods increased from about 200 in the 20th week to a maximum of 575 movements in the 32nd week. Weekly fetal movement counts then declined to an average of 282 at 40 week. Normal weekly counts of fetal movements ranged between 50 and 950, with large daily variations that included counts as low as 4 to 10 per 12 hours/day in normal pregnancies.

Since **Sadovsky and yaffe (1973)** described seven case reports of pregnancies with decreased fetal activity that preceded fetal death, there have been various methods described to discuss and quantify fetal movements for the purpose of prognosticating fetal well-being. Methods used to document fetal activity include use of tocodynamometer, visualization with real-time ultrasound and maternal subjective perceptions.

Rayburn (1980) found that 80% of all movements seen during ultrasonic monitoring were perceived by the mother. In contrast, Johnson and colleagues (1992) reported that beyond 36 weeks, mothers perceived only 16 per cent of fetal body movements recorded by a Doppler device. Fetal motions lasting more than 20 seconds were identified more accurately by the mother than shorter episodes of fetal activity.

Rayburn (1982) defined reassuring maternally perceived fetal activity as an average of four or more movements per hour when counting was performed at least 1 hour per day. Three or fewer movements per hour for two consecutive days was defined to be abnormal.

In earlier days when stethoscope was not in much use fetal movement count was one of the methods to assess fetal well being in ante partum period. Any of the following method can be used.

Cardiff "Count 10" Formula- Patient counts fetal movements starting at 9 a.m. The count comes to an end as soon as 10 movements perceived. She is instructed to report the physician, if: (i) Less than 10 movements occur after 12 hours on successive days; or (ii) no movement is perceived even after 12 hours in a single day.

Daily Fetal Movement Count (DFMC)- Three counts each of one hour duration (morning, noon and evening) are recommended. The total counts multiplied by four gives daily (12 hour) foetal movement count. If there is diminution of the number of "kicks" to less than 10 in 12 hours, it indicates failing placental function.

The count is performed 3 or 4 times a week. Loss of fetal movements is commonly followed by disappearance of FHR within next 24 hours.

III. INTERMITTENT AUSCULTATION

For long intermittent auscultation (every 15 minute in 1st stage and every 5 minute during second stage) remained an acceptable method of fetal monitoring in all patients. But later it was realized that this method is subjected to considerable observer variability apart from this during auscultation baseline heart rate can be measured but other features such as

baseline variability, acceleration and deceleration are difficult to quantify. So need for electronic fetal monitoring evolved.

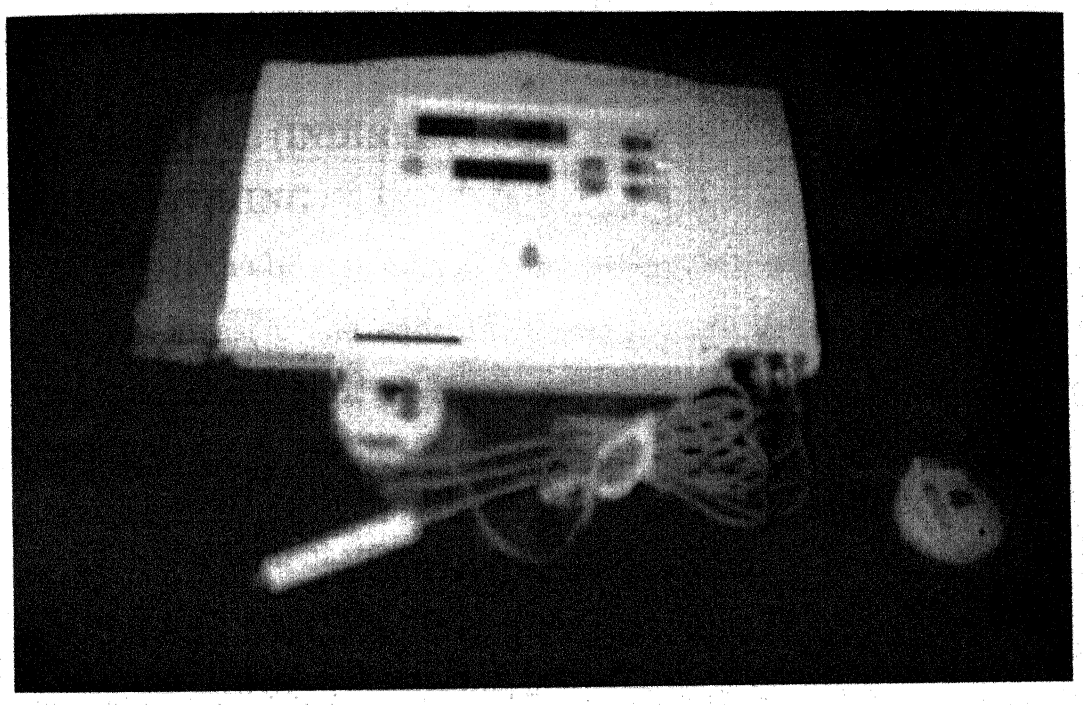
IV. ELECTRONIC FETAL MONITORING

In 1953, Gunn and Wood reported – "The amplification and recording of fetal heart sounts" in "The Proceedings of the Royal Society of Medicine". In 1958, Hon pioneered electronic fetal monitoring in USA. Caldeyero-Barecca and Hamma reported their observations on various heart rate patterns in fetal distress. This set the scene for production of different kinds of fetal monitors.

Continuous electronic fetal monitoring was a marvellous invention introduced in the obstetrical practice in 1960s. No longer was the perception of fetal distress limited to heart sounds, the continuous graph paper portrayal of the fetal heart pattern was potentially diagnostic in assessing patho-physiological events affecting the fetus.

The first report on clinical application of electronic fetal monitoring in USA was from Paul and Hon in 1970. They carried out their study among the high-risk patients and concluded that electronic fetal monitoring was beneficial in complicated pregnancies because of finding of lesser number of depressed babies in monitored groups.

Initial prospective randomized studies of electronic fetal monitoring comparing it with intermittent ausculation were conflicting with Renow et al. (1976) reporting a significant reduction in morbidity with its use- while Haverkamp et al. (1979) found no benefit and indeed reported dramatic and apparently unnecessary increase in caesarian section rate resulting from its use.



FETAL MONITOR WITH ITS ATTACHMENTS

National Maternity Hospital, Dublin was the site of the largest randomized study of electronic monitoring (Mac Donald & Coworkers). Incidence of forceps delivery was more in electronically monitored group but caesarean delivery rates did not differ. No differences were found in the incidence of intrapartum still births or neonatal deaths.

Luthy and Coworkers (1987) studied the effects of electronic monitoring on neurobehabiourable development of few birth weight infants. Electronic monitoring can be done internally as well as externally.

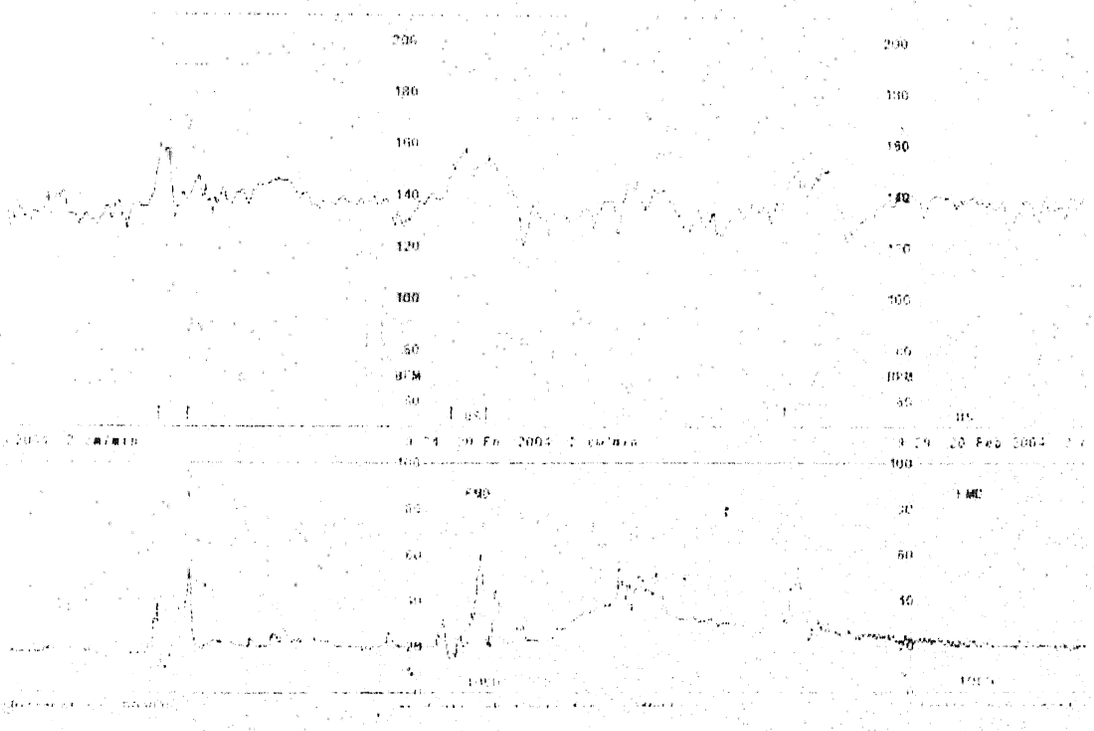
EXTERNAL (INDIRECT) ELECTRONIC FETAL HEART RATE MONITORING

It can be applied both in anterpartum as well as intrapartum period. It is non-invasive and widely acceptable. Only drawback is that it does not provide precision of fetal heart measurement or the quantification of uterine pressure afforded by internal monitoring.

The fetal heart may be detected in a number of ways through maternal abdominal wall. The easiest technique employs the ultrasound Doppler principle. Ultrasonic waves undergo shift in frequency as they are reflected from moving heart valves and from blood ejected in pulsatile fashion during systole.

Unit consists of a transducer that emits ultrasound and a sensor to detect a shift in frequency of reflected sound. The transducer is placed on maternal abdomen at a site where fetal heart action is best detected. A coupling gel must be applied because air conducts ultrasound poorly. The device is held in a position by a belt. Care should be taken that maternal aortic pulsations are not confused with fetal cardiac motion.

Ultrasound Doppler signals are edited technically before fetal heart rate data printed onto the bedside monitor tracing paper.



FHR TRACING SHOWING NORMAL BASELINE, NORMAL BEAT TO BEAT VARIABILITY AND ACCELERATION WITH FETAL MOVEMENT

FETAL HEART RATE PATTERNS

Interpretation of electronic fetal heart rate data is based upon visual pattern of heart rate as portrayed on chart recorder graph paper.

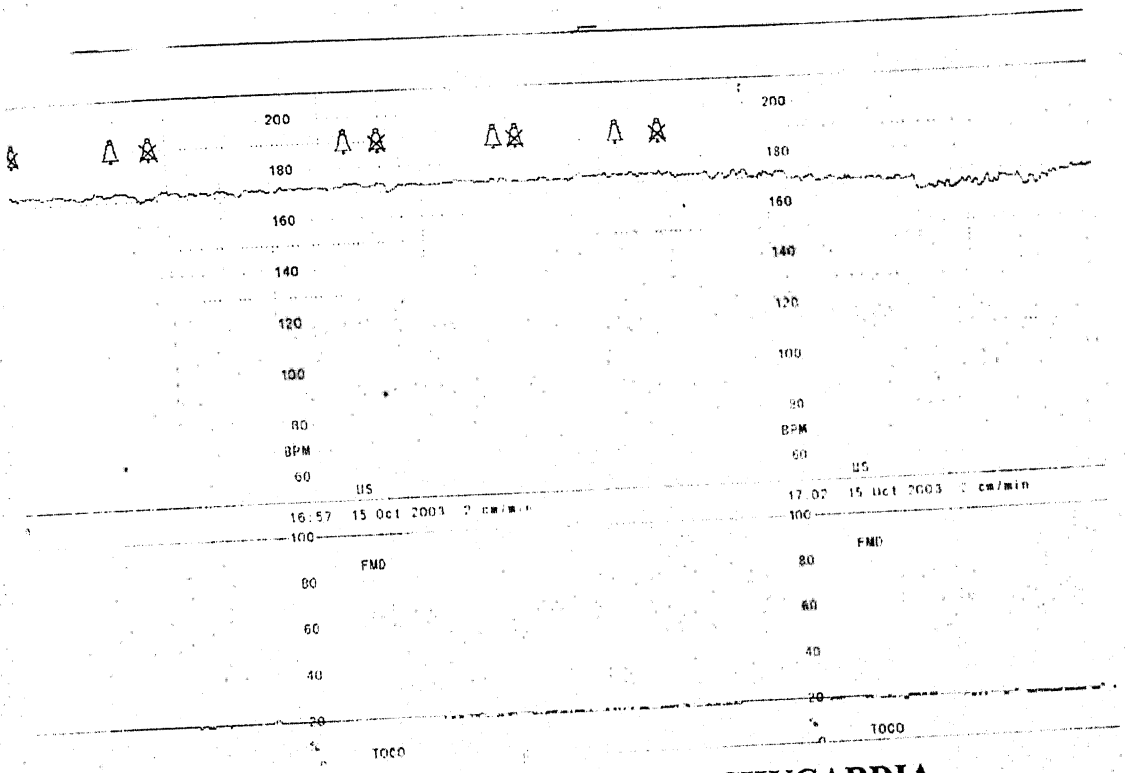
Base line fetal heart activity : Descriptive characteristics of base line fetal heart activity include rate, beat to beat variability, fetal arrhythmia and distinct patterns like sinusoidal and saltatory heart patterns.

Rate : With increasing fetal maturation, the heart rate decreases.

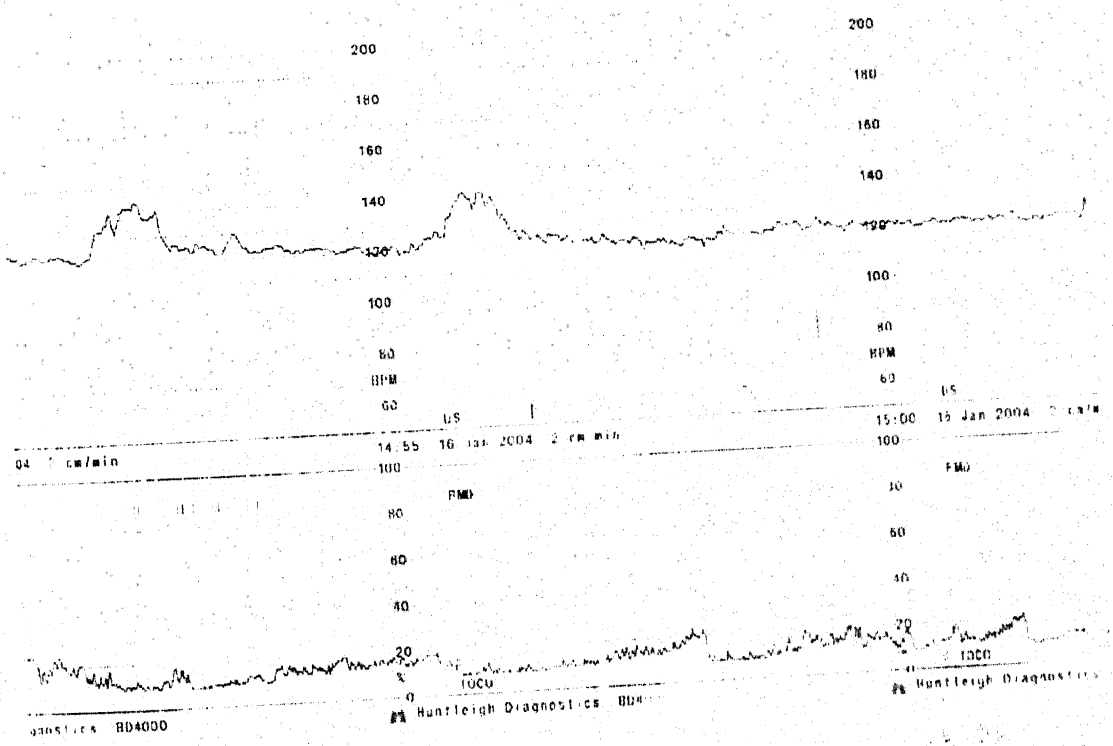
Pillai and James longitudinally studied fetal heart rate characteristics in 43 normal human pregnancies from 16 to 40 weeks. The baseline fetal heart rate decreased an average of 24 beats/minutes between 6 weeks and terms or approximately 1 beat/minute per week. At 16 weeks the average baseline rate was about 160-beats/ minute, which decreases to 40/minute at 40 weeks. It is postulated that this normal slowing of the fetal heart rate corresponds to maturation of parasympathetic (Vagal) heart control.

During third trimester, the normal average baseline heart rate is generally accepted to be between 120 and 160/minute. The average fetal heart rate is considered to be the result of tonic balance between accelerator and decelerator influences on pacemaker cells, sympathetic system is the accelerator influences and the parasumpathetic system is the decelerator factor mediated via vagal slowing of the heart rate. Heart rate is also under control of arterial chemoreceptors such that both hypoxia and hypercapnia can modulate rate. More severe and prolonged hypoxia with a rising blood lactate level and sever metabolic acedemia, includes a prolonged fall of heart rate due to direct effects on the myocardium.

Bradycardia : Bradycardia is a baseline heart rate of under 120 beats/minutes that lasts 15 minute or longer. However, a rate between 100



FHR TRACING SHOWING TACHYCARDIA



FHR TRACING SHOWING BRADYCARDIA

and 119 beats/minute in the absence of other changes is usually not considered to represent fetal compromise.

Moderate bradycardias are defined as 80-100 beats/minute and sever bradycardias are less than 80 beats/ minute for 3 minutes or longer.

Mild bradycardia without deceleration or acceleration is not necessarily evidence for fetal compromise.

Other causes of fetal bradycardia include congenital heart block and serious fetal compromise.

Other non periodic but sudden slowing of fetal heart rate are often termed fetal "bradycardias". Some causes include uterine hyperactivity paracervical or conduction analgesia, pelvic examination, presumably due to manual fetal head compression and prolapse, uterine rupture, placental abruption, maternal hypoperfusion (e.g. Supine hypotension syndrome or haemorrhage due to trauma) and maternal hypoxia (e.g. eclampsia).

Tachycardia : Tachycardia is considered by most as mild if the baseline rate is between 161 and 180 beats/minute and servee if 181 or more. Most common explanation for fetal tachycardia is maternal fever from amnionitis, although fever from any source can increase baseline fetal heart rate. Fetal tachycardia caused by maternal infection is typically not associated with fetal compromise unless there are associated periodic heart rate changes or fetal sepsis.

Other causes of fetal tachycardia inclue fetal compromise, cardiac arrhythmias and administration of parasympathetic (atropine) or sympathomimetic (terbutaline) drugs to the mother.

Key feature to distinguish fetal compromise in association with tachycardia seems to be concomitant heart rate decelerations.

Beat to Beat Variability : Baseline fetal heart rate variability is an important index of cardiovascular function and appears to be regulated largely by the autonomic nervous system.

The baseline rate normally exhibits an oscillating form, reflective of beat to beat changes in rate, which give it varying degrees of irregularity or variability when printed on graph paper. Variability is further divide as :

Short term Variability : Reflects instantaneous change in fetal heart rate from one beat (or R wave) to the next. Variability is a measure of the time interval between cardiac systoles.

It can most reliably be determined to be normally present only when electrocardiac cycles are measured directly with a scalp electrode.

External doppler ultrasound recording methods can create artificial "normal" variability. Conversely, absence of variability during external doppler fetal heart recording can suggest loss of beat to beat variability.

Long term Variability : is used to describe oscillatory changes that occur during course of 1 minute and result in the waviness of the baseline.

Normal frequency of such waves is 3-5 cycles/minute.

Decreased beat to beat variability is diagnosed when the fetal heart rate baseline is flat or nearly flat with absent short term variability and fewer than two cyclic changes per minute of long term variability.

Several physiological and pathological processes can affect or interfere with beat to beat variability. Dawes and coworkers described increased beat to beat variability during fetal breathing. In healthy infants short term variability is attributable to respiratory sinus arrhythmia.

Moreover, this respiratory variability can be reduced by asphyxia. Fetal body movements affect variability.

VanGejn and coworkers (1980) analysed electro-encephalographic data in healthy term infants and observed 30 to 70 minute sleep cycles corresponding to fetal physical inactivity. Pillai and James (1990) reported increased baseline variability with advancing gestation. Upto 30 weeks baseline characteristic were similar during both fetal rest and activity. After 30 weeks fetal inactivity was associated with diminished baseline variability and conversely, variability was increased during fetal activity.

It is recognized that baseline fetal heart rate becomes more physiologically fixed (less variable) as the rate increases. Conversely, there is more instability or variability of the baseline at lower heart rates. This phenomenon presumably reflects cardiovascular physiological wandering as beat to beat intervals shorten due to increasing heart rate. It is generally believed that all these physiological processes modulate variability via the autonomic nervous system **(Renou and coworkers, 1969).**

Diminished beat to beat variability can be an omnious sign indicating a seriously compromised fetus (Paul and coworkers, 1975) reported that loss of variability in combination with decleration was associated with fetal acidemia. They defined decreased variability as 5 or fewer beats/minute.

Chaffin and coworkers (1996) recently provided evidence that decreased variability seen with chronic fetal asphyxia is not the result of acedemia alone. Severe maternal acidemia can also cause decreased fetal heart rate beat to beat variability as in mother with diabetic ketoacidosis.

Although loss of variability and its ominous significance are concepts familiar to most obstetricians, mild degrees of fetal hypoxemia

during human labour have been reported to increase variability at least at the onset of the hypoxic episode.

According to Dawes (1985), it seems probable that the loss of variability is a result of metabolic acidemia that causes depression of fetal brainstem or the heart itself. Thus, diminished beat to beat variability, when a reflection of compromised fetal condition, likely reflects academia rather than hypoxia.

A common cause of diminished beat to beat variability is analgesic drugs given during labour. A large variety of nervous system depressant drugs can cause transient diminished beat to beat variability. Included are narcotics, barbiturates, phenothiazines (e.g. promethazine), tranquilizers (e.g. diazepam) and general anaesthetics.

It is generally believed that reduced baseline heart rate variability is the single most reliable sign of fetal compromise. For example, Smith and coworkers (1988) performed a computerized analysis of beat to beat variability in growth restricted fetuses before labour. They observed that diminished beat to beat variability (4.2 beats/minute or less) that was maintained for 1 hour is diagnostic of developing acidemia and imminent fetal death.

Cardiac Arrhythmia

When fetal cardiac arrhythmias are first suspected using electronic monitoring, findings can include baseline bradycardia, tachycardia or most commonly abrupt baseline spiking. Intermittent baseline bradycardia is frequently due to arrythmia and can only be accomplished, practically speaking, when scalp electrodes are used.

Most supraventricular arrhythmias are of little fetal significance during labour unless there is coexistent heart failure as evidenced by hydrops.

Other than ectopic systoles, which are as common in fetuses as in adults, ventricular arrhythmias are unusual in utero. Conduction defects most commonly complete atrioventricular block; are usually found in association with connective tissue diseases.

The clinical significance of intrapartum fetal arrhythmia continues to be a complex problem. Most are of little consequence during labour when there is no evidence of fetal hydrops. However, such arrhythmias impair interpretation of intrapartum heart rate tracings.

Sinusoidal Heart Rates

Discovery of sinusoidal heart rates is attributable to Kubli and coworkers and Manseau and colleagues (1972). A true sinusoidal pattern may be observed with serious fetal anemia whether from D-isoimmunization, ruptured vasa previa, fetomaternal haemorrhage or twin to twin transfusion. Insignificant sinusoidal patterns have been reported following administration of nepridine, morphine alphapoodine, butorphanol (Angel, 1984; Egley, 1991; Epstein, 1982 and their associates). The pattern has also been described with amnionitis, fetal distress and umbilical cord occlusion (Murphy and associates, 1991). Young and coworkers (1980a) and Johnson and coworkers concluded that intrapartun sinusoidal fetal heart patterns were not usually associated with fetal compromise.

Madaulou and Freeman (1982) based on their extensive review, proposed adoption of a strict definition – (1) stable baseline heart rate of 120-160 beats/minute with regular oscillations; (2) amplitude of 5 to 15 beats/minute (rarely greater); (3) frequency of 2 to 5 cycles/minute long term

variability; (4) fixed or flat short- term variability; (5) oscillation of sinusoidal wave form above or below a baseline; (6) absence of accelerations.

Other investigators have proposed-a classification of sinusoidal heart rate pattern into mild – amplitude 5-15 beats/minute; intermediate – 16-24 beats/minute and major 25 beats or more, to quantify fetal risk (Murphy and colleagues, 1991; Neesham and coworkers, 1993).

Some investigators have defined intrapartum sine wave like baseline variation with periods of acceleration as pseudo-sinusoidal.

The pathophysiology of sinusoidal pattern is unclear, in part due to various definitions. There seems to be general agreement that antepartum sine wave baseline undulation portends severe fetal anemia, however few D-isoimmunized fetuses develop this pattern (Nicolaides, 1989). The sinusoidal heart rate did not appear to be under the influence of the α or β sympathetic systems.

Periodic Fetal Heart Rate

The periodic fetal heart rate refers to deviations from baseline that are related to uterine contractions. Accelerations refers to an increase in fetal heart rate above baseline rate and deceleration to a decrease below baseline rate. The most common used system in the United States is based on the timing of deceleration in relation to contraction, thus early, late or variable in onset compared with the corresponding uterine contraction. The waveform of these deceleration is significant for pattern recognition in early and late decelerations the slope of fetal heart rate change is gradual, resulting in curvilinear and uniform or symmetrical waveform. With variable deceleration, the slope of fetal heart rate change is abrupt and erratic giving the wave form a jagged appearance.

Another system used for description of deceleration is based on pathophysiological events. In this system early decelerations are termed head compression, late decelerations are termed uteroplacental insufficiency and variable decelerations become cord compression patterns.

Accelerations

An acceleration is an increase in fetal heart of at least 15 beats/minute usually of 15 to 20 seconds duration. According to Freeman and coworkers (1991) accelerations occur most commonly antepartum in early labour and in association with variable deceleration.

Proposed explanations, for intrapartum acceleration include fetal movement, stimulation by uterine contractions, umbilical cord occlusion and fetal stimulation during pelvic examination.

Fetal scalp blood sampling and acoustic stimulation both incite fetal heart rate acceleration (Clark and coworkers, 1982). Finally acceleration can also occur during labour without any apparent stimulus. Indeed, accelerations are common in labour and nearly always associated with fetal movements. These accelerations are virtually always reassuring and almost always confirm that the fetus is not acidotic at that time.

Accelerations seem to have the same physiological explanations as beat to beat variability in that they represent intact neurohormonal cardiovascular control mechanisms linked fetal behavioural states. Accelerations during the first and/or last 30 minutes was a favourable sign for fetal well being. The absence of fetal heart accelerations during labour, however is not necessarily an unfavourable sign unless coincidental with other non-reassuring changes.

Early Deceleration

Early deceleration of fetal heart rate was first described by Hon (1958). He observed that there was a drop in heart rate with uterine contractions and that this was related to cervical dilatation. He considered these physiological. Compressing, the fetal head produced variable type decelerations in 18 of 19 attempts (Ball and Parer, 1992). Similar decelerations were elicited by locking of forceps and initiation of traction.

Freeman and co-authors (1991) defined early decelerations as those generally seen in active labour between 4 and 7 cm dilatation. In their definition, the degree of deceleration is generally proportional to the contraction strength and rarely falls below 100 to 110 beats/minute or 20 to 30 beats/minute below baseline. Importantly early decelerations are not associated with fetal hypoxia, academia or low apgar scores.

Head compression probably causes vagal nerve activation due to dural stimulation that mediates heart rate deceleration (Paul and coworkers, 1964).

Late Deceleration

The fetal heart rate response to uterine contractions can be an index of either uterine perfusion or placental function. Because even normal uterine contractions interdict uteroplacental perfusion it was anticipated very early in the history of electronic monitoring that the stress of contractions could become the basis for a clinical test for placental respiratory function.

A late deceleration is a symmetrical decrease in fetal heart rate beginning at or after the peak of the contraction and returning to baseline only after the contraction has ended (American Colleage of Obstetrics and Gynaecologists, 1995b). Late decelerations are uniform in shape and typically begin 30 seconds or more after the onset of the contraction.

Nadir of deceleration is after the contraction and return to baseline is well after the contraction is over. Descent and return of the fetal heart rate are gradual and smooth. The magnitude of late decelerations reportedly is rarely more than 30-40 beats/minute below baseline and typically not more than 10 to 90 beats/minute in intensity. Late decelerations are usually not accompanied by accelerations.

Myers and associates (1973) studied monkeys in which they compromised uteroplacental perfusion by lowering maternal aortic blood pressure. The time interval of lag periods from the onset of a late deceleration was directly related to basal fetal oxygenation. They demonstrated that the length of the lag phase was predictive of the fetal PO2 but not fetal pH.

Murata and associates (1982) also showed that a late deceleration was the first fetal heart rate consequence of uteroplacental induced hypoxia. Variability of baseline heart rate disappeared as acidemia developed.

The precise pathophysiological mechanisms whereby fetal hypoxia is translated into fetal heart rate effects are unclear. Harris and colleagues (1982) studied mechanisms of late decelerations in fetal lambs and concluded that there are two pathophysiological pathways: (1) chemoreceptor-mediated vagal reflex and; (2) hypoxic myocardial depression. They observed that it often is noticed clinically that sudden maternal hypotensionor uterine hyperstimulation with oxytocin in a previously normal fetus results in late decelerations with retention of beat to beat variability. They postulated that the chemoreceptor vagal nerve reflex mechanism is involved in such circumstances. In contrast, with prolonged fetal hypoxia, late deceleration is mediated via direct myocardial depression and baseline variability is absent.

The method for inducing uteroplacental insufficiency and late decelerations in the animal experiments cited was interference with maternal aortic blood flow. Generally, any process that causes maternal hypotension, excessive uterine activity, or placental dysfunction can induce late decelerations. The two most common causes are hypotension from epidural analgesia and uterine hyperactivity due to oxytocin stimulation. Maternal diseases such as hypertension, diabetes and collagen-vascular disorders can cause chronic placental dysfunction. A rare cause is severe chronic anemia without hypovolemia. Placental abruption can cause acute and severe late decelerations.

Variable Decelerations :

Most common deceleration encountered during labor are variable decelerations attributed to umbilical cord occlusion. Release of amniotic fluid and fetal descent during parturition are conducive to umbilical cord entrapment. One fourth of fetuses have one or more loops of cord wound round the neck. Similarly short cord (less than 35 cm) and long (more than 80 cm) cords are found in 6% of births and are associated with variable decelerations (Rayburn and associates, 1981). Melchor and Bernard (1985) identified variable decelerations in 40% of over 7000 monitor tracing when labour has progressed to 5 cm dilatation and in 83% by end of the first stage.

Very early in the development of electronic monitoring, Hon (1959) tested the effects of umblical cord compression on fetal heart rate. Similar complete occlusion of the umblical cord in experimental animal produces abrupt, jugged appearing deceleration of the fetal heart rate.

Lee and coworkers (1975) proposed that the variation of variable deceleration was caused by differing degrees of partial cord occlusion. In this physiological scheme, occlusion of only vein reduces fetal blood return

thereby triggering a baroreceptor-mediated acceleration. Subsequent complete occlusion results in fetal systemic hypertension due to obstruction of umbilical artery flow. This stimulates a baroreceptor-mediated deceleration. Presumably, the aftercoming shoulder of acceleration represents the same events occurring in reverse.

Ball and Parer (1992) concluded that variable decelerations are vagally mediated and that the vagal response may be due to chemoreceptor or baroreceptor activity or both. Partial or complete cord occlusion (baroreceptor) produces afterload increase, hypertension and decrease in fetal arterial oxygen content (chemoreceptor), both of which result in vagal activity leading to deceleration.

Variable decelerations represent fetal heart rate reflexes that either blood pressure changes due to interruption of umbilical flow or changes in oxygeneation. The frequency and inevitability of cord occlusion has undoubdtly provided the fetus with these physiological mechanisms as a means of coping. Hence, we have elected to term these reflexes "physiological" rather than patho-physiological. The American College of Obstetricians and Gynecologists (1995b) has defined significant as those decreasing to less than 70 beats/sec and lasting more than 60 seconds.

Other fetal heart rate patterns have been associated with umbilical cord compression. Saltatory baseline heart rate was first described by Hammacher and coworkers (1968) and linked to umbilical cord complications during labour. The pattern is considered due to rapidly recurring couplets of acceleration and deceleration causing relatively large oscillations of the baseline heart rate.

Goldkrand and Speichinger (1975) described a mixed cord compression pattern consisting of an acceleration immediately followed by a

deceleration associated with abnormal cord positions at delivery. Aladjem and associates (1977) subsequently termed this acceleration – deceleration combination the lambda pattern and attributed it to fetal movement. Brubaker and Garite (1988) identified the lambda pattern in 4% of labours and concluded that it was not associated with adverse outcomes.

Prolonged Decelerations : Prolonged decelerations are defined as isolated decelerations lasting more than 60-90 seconds (Freeman and Coauthors, 1991). Because baseline rate refers to a baseline lasting 15 minute or longer, and prolonged decelerations would be those lasting more than 60 to 90 seconds and less than 15 minutes.

Prolonged decelerations are difficult to interpret because they are seen in many different clinical situations. Some of the common causes include cervical examination, uterine hyperactivity, cord enlargement and maternal supine hypotension.

In a study by Tejani and associates (1975) the longest prolonged deceleration was 12 minutes.

Other causes of prolonged deceleration include epidural, spinal or paracervical analgesia, maternal hypoperfusion or hypoxia due to any cause, placental abruption, umbilical cord knots or prolapse, maternal seizures including eclampsia and epilepsy, application of a fetal scalp electrode, impending birth or even maternal valsalva maneuver.

The placenta is very effective in resuscitating the fetus if the original insult dose not recur immediately.

Non Stress Test

In 1975 Freeman and colleagues introduced non stress test to describe fetal heart rate acceleration in response to fetal movement as a sign of fetal health. It is the test used for antepartum evaluation of fetal well being. The rationale underlying this test is the presence of spontaneous fetal heart rate acceleration associated with fetal movements.

The non stress test based on the heart rate of the fetus who is not aciodotic as a result of hypoxia or neurological depression will temporarily accelerate in the response to fetal movement.

The definition currently recommended by the American College of Obstetrics & Gynecologists (1994) is 2 or more acceleration of 15 beats/ minute or more, each lasting 15 seconds or more and occurring within 20 minutes of the beginning of the test. It is also recommended that acceleration with or without fetal movements be accepted and that a 40 minute or longer tracing (to account for fetal sleep cycle) should be performed before concluding that there was insignificant fetal reactivity. The variables evaluated are :

(1) Baseline fetal heart rate
(2) Variability of fetal heart rate
(3) Presence or absence of acceleration
(4) Presence or absence of deceleration

On the basis of these criteria non-stress test is of following type :

(1) Normal / Reactive / Reassuring
(2) Pathological / Nonrecative / Ominous
(3) Suspicious / Equivocl.

Normal / Reassuring / Reactive :

- Baseline heart rate 110-150 bpm.
- Baseline variability 10-25 bpm.
- Fetal movements at least two.
- Presence of accelerations of at least >15 bpm lasting >15 sec.
- Absence of decelerations.

Suspicious / Equivocal :

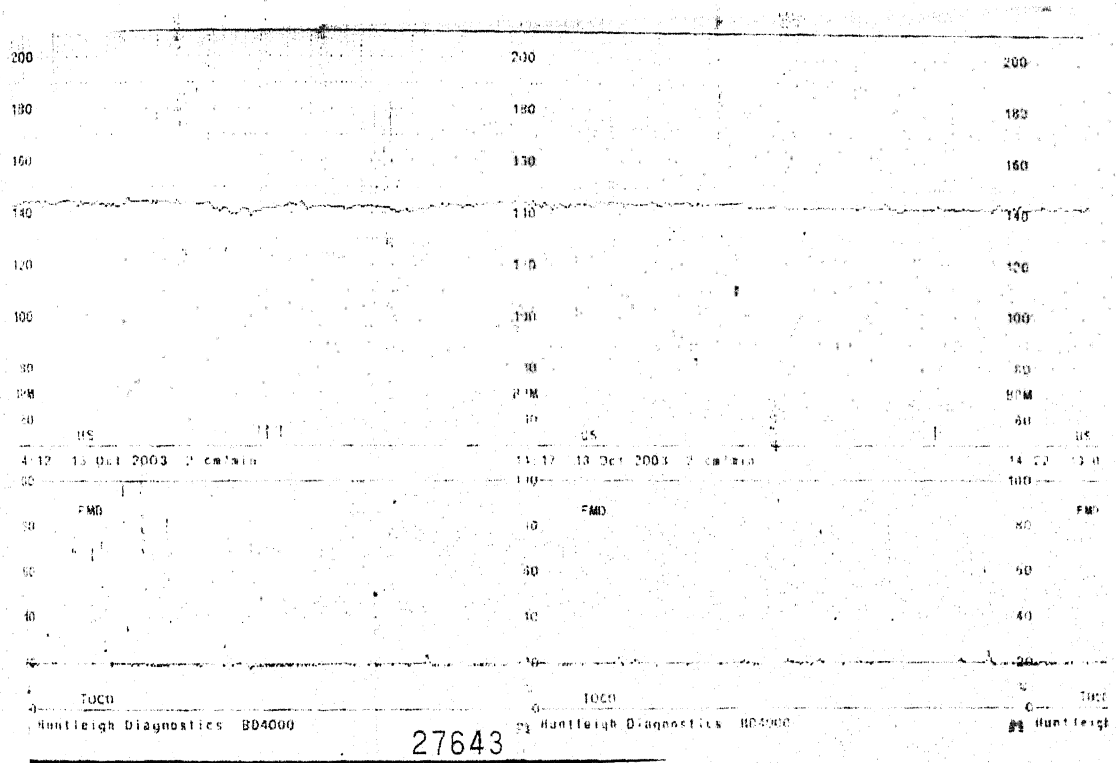
- Baseline heart rate 150-170 bpm or 100-110 bpm.
- Reduced baseline variability 5-10 bpm for >20 min with normal baseline and no deceleration.
- Fetal movements <2.
- Absence of acceleration >40 min.
- Variable decelerations <60 bpm for <60 sec.

Non reactive / Pathologic / Ominous

- Baseline heart rate <100 bpm or >170 bpm.
- Silent pattern <5 bpm variability for >40 minutes.
- Fetal movement absent.
- Deceleration variable >60 bpm lasting for >60 sec or repetitive late decelerations.

Freeman (1975) and Lee & colleauges (1975) introduced the non stress test to describe fetal heart rate acceleration in response to fetal movement as a sign of fetal health.

Brown and Patrik (1981) considered that a longer duration of non-stress testing might increase the positive value of an abnormal or non-reactive test, because healthy fetuses may not move for the period of upto 75 minutes. They concluded that either the test become reactive during a period



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FHR TRACING WITH NORMAL BASELINE RATE BUT SILENT PATTERN
(i.e. beat to beat variability <5bpm)

of time upto 80 minutes or that the test remained non-reactive for 120 minutes indicating that fetus was very ill.

In 1988 Smith & colleagues observed a decrease in the number of acceleration in preterm human fetuses subsequently found to have lower umbilical artery blood PO2 values compared with those fetuses who had normal fetal heart rate characteristics.

Gestational age also is a factor influencing acceleration or reactivity of fetal heart rate. Pillai & James (1990) studied the development of fetal heart rate acceleration pattern during normal pregnancy. The percentage of body movements accompanied by acceleration and the amplitude of these accelerations increase with the gestational age.

As initially devised, non-stress testing involved the detection of acceleration associated with document fetal movement. Subsequent studies however demonstrated that appropriate accelerations are predictive of fetal well being regardless or presence of maternally perceived fetal movements (Devac & associates), 1994; Stanco & colleagues, 1983.

Indeed a recent investigator using Doppler detected fetal body movement during-stress testing suggested that this technique may allow the clinical to avoid inappropriate diagnosis of fetal compromise when the non-stress test is non-reactive (Devoe & cowerkers, 1994).

Although a normal number and a amplitude of accelerations to reflect fetal well being but "insufficient acceleration" does not invariably fetal compromise.

Indeed some investigators have reported false positive non-stress test ratio in excess of 90 percent when fetal heart rate acceleration was considered insufficient (Devoe & colleagues, 1986).

Not only are there different definitions of normal non-stress results but the reproducibility and interpretation is also problematic. For example Hage (1985) mailed non-stress test, blinded to specific patient clinical data, to a national sample of obstetrician for the interpretation. He concluded that although non-stress testing is very popular with subjected visual reliability of test interpretation needs improvement. Such problems with subjected visual interpretation

Visser and associates (1980) described a 'terminal cardiotocogram' which included :

(i). Baseline oscillations of <5 beats/minutes;

(ii). Absent accelerations;

(iii). Late deceleration with spontaneous uterine concentration.

Devoc & co-workers (1985) concluded that non-reactive non-stress test for 90 minutes was almost invariably (93%) associated with significant perinatal pathology.

Hoskins & associates (1991) attempted to refine interpretation of non-stress tests showing variable deceleration by ultrasonic estimation of amniotic fluid volume. The incidence of lower segment caesarean section for intrapartum fetal distress progressively increased coincidently with the severity of variable deceleration and diminished amniotic fluid volume. Fetal distress in labour, also frequently developed in those pregnancies with variable deceleration but with normal amniotic fluid.

Noren H & colleagues (2003) evaluated cardiotocography plus automatic ST analysis of the fetal electrocardiography. It has been shown recently to reduce both the operative delivery rate for fetal distress and the cord artery metabolic aciodosis rate. 4966 term fetuses were included in the trial. Of the 29 fetuses with adverse/complicated neonatal outcome, 22

fetuses had cardiotocography plus ST paterns that indicated a need for intervention, according to the cardiotocography plus St clinical guidelines.

Williams KP & colleagues (2003) evaluated the ability of two different modes of antepartum fetal testins to screen for the presence of peripartum morbidity, as measured by the cesarean delivery rate for fetal distress in labour. One thousand three hundred sixty patients were assigned randomly to groups in the study. They concluded that umbilical artery Doppler as a screening test for fetal well being in a high-risk population was associated with a decreased incidence of caesarean delivery for fetal distress compared to the non stress testing, with no increase in neonatal morbidity.

INTERNAL FETAL MONITORING :

In internal fetal monitoring fetal heart rate may be measured by attaching a bipolar spiral electrode directly to the fetus.

The wire spiral electrode penetrates fetal scalp and the second pole is the metal wing on the electrode panel. Vaginal fluid create a saline electrical bridge that completes the circuit and permits measurement of the voltage differences between the two poles. The electrical fetal cardiac signal (P wave, QRS complex and T wave) is amplified and fed into cardiotocometer for heart rate calculation. The peak R wave voltage is the portion of the fetal electrocardiogram most reliably detected. The two wire of bipolar electrodes are attached to material thigh to a reference electrode to eliminate electrical interference. Time (t) in milli seconds between fetal R waves is fed into a carditocometer where a new fetal heart rate is set with arrival of each new R wave. A premature atrial contraction is computed as a heart acceleration because the internal t_2 is shorter than t_1.

The phenomenon of continuous R wave to R wave fetal heart rate computation is known as "beat to beat" variability.

Maternal cardiac complexes are also detected by fetal scalp electrode but in live fetus this low amplitude maternal ECG signal is detected but masked by the noise of the fatal ECG.

Internal monitoring has other disadvantages also – being an invasive process it can cause infection and scalp abscess. It can only be applied after the rupture of membranes.

Every fetus has a potential risk of intrapartum hypoxia or birth injury and an optimal outcome can be concluded only at the end of labour or occasionally, much later. Therefore, every fetus deserves intrapartum monitoring. Most patients with uncomplicated low risk pregnancies do not need electronic fetal monitoring and may be effectively monitored by traditional method of intermittent auscultation of the fetal heart. Electronic fetal monitoring can be applied to patients with one or more high risk factors.

(a). Risk arising from problems of the fetus.

1. Intrauterine growth retardation
2. Post term
3. Preterm
4. Oligohydraminics
5. Breech presentation
6. Rhesus isoimmunization

(b). Risk arising from maternal medical problems.

1. Hypertension
2. Diabetes
3. Renal disease
4. Severe anaemia
5. Heart disease
6. Hyperthyrodism

(c). Risks arising from problems of labour

1. Induced labour
2. Augmented labour
3. Prolonged labour
4. Previous caesarian

(d). Suspected fetal distress in labour

1. Meconium stained amniotic fluid
2. Abnormal / suspicious admission test
3. Suspicious FHR on auscultation

Intermittent auscultation Vs. continuous electronic fetal monitoring :

Undoubtedly continuous electronic fetal monitoring provides more information regarding the FHR than intermittent auscultation with a fetal stethoscope. Listening to the fetal heart rate for a minute every 15 minutes samples the FHR for only about 7% of the total time in labour (S.Arulkumaran, S.S. Ratnam). In addition it provides very little information about the relationship between FHR changes and uterine contractions, or about FHR variability.

The value of continuous fetal heart rate monitoring in labour in low risk gravidas remains unproven. When the results of nine prospective randomized controlled trials of the use of continuous FHR monitoring in labour were pooled for systematic meta-analysis, the results showed that continuous EFM caused higher rates of medical intervention; both cesarean section and instrumental delivery, without any accompanying decrease in perinatal mortality (Shy et al. 1990). When compared with intermittent auscultation alone continuous FHR monitoring doubled rates of caesarean section for "fetal distress" even when it is complemented by selective fetal

blood sampling (FBS) and pH estimation, the rate was quadrupled when EFM was use alone (Neilson, 1993). Although in the largest randomized trial involving nearly 13,000 women, carried out in Dublin, a 55% decline in the incidence of neonatal convulsions was noted in the group who had EFM.

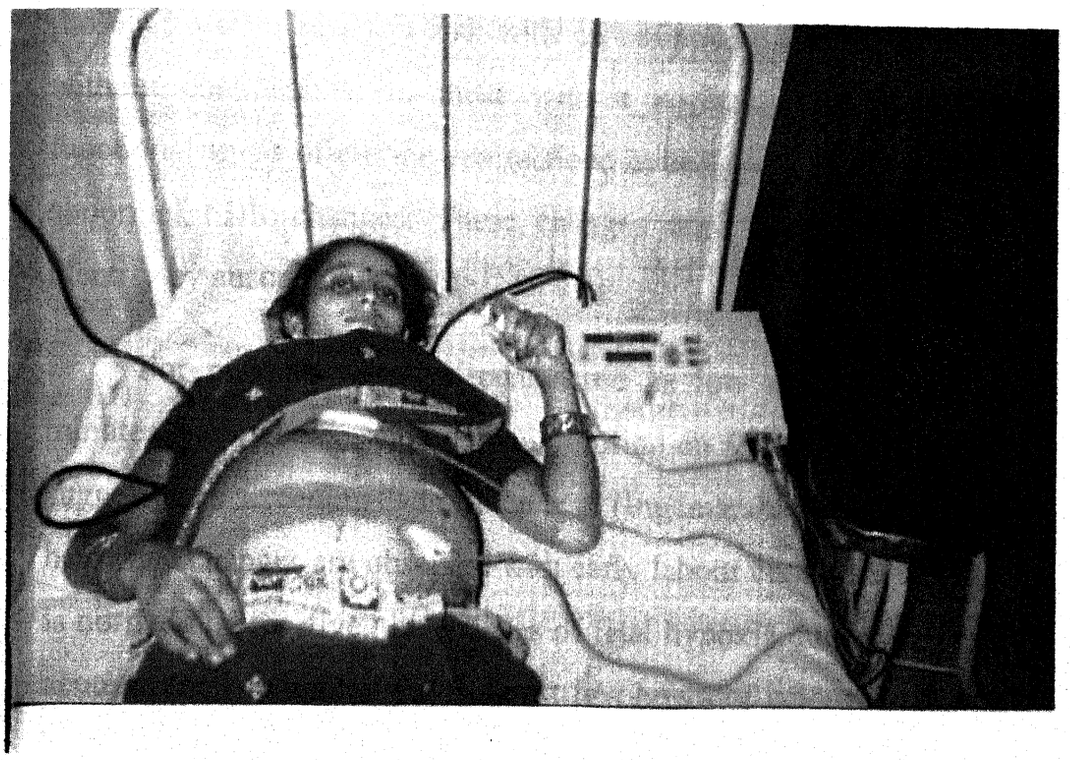
Andreas Herbst & Ingeman Ingeamarsson (Aug 1994) conducted a prospective randomized study on 4044 patients in labour & concluded that intermittent use of EFM at regular intervals with stethescope auscultation in between appears to be as safe as continuous EFM in low risk women. Further, stratified analysis showed that the reduction in neonatal convulsion occurred very largely in those women labours had lasted more than 5 hour (Mac Donald et al, 1985). This suggests that regular intermittent auscultation is sufficient and an acceptable form of monitoring in low risk mothers who progress to delivery within five hours. Continuous FHR monitoring is more appropriate in more complicated labours, such as those that are prolonged, augmented or induced, those with thick meconium stained amniotic fluid, and those with a known or suspected growth retarded or preterm fetus.

For the proper allocation of available resources CTG machines and manpower, it is important to identify the mother who is genuinely "low risk" for the development of intrapartum hypoxia.

ADMISSION TEST :

In view of non-applicability of continuous monitoring to all patients and other drawbacks, there has been a search for newer techniques which is non-invasive and less costly.

Admission test is one such technique by which by taking a single trace at the time of admission in early labour two things can be predicted. One is the asphyxia which is already present at the time of admission and other is asphyxia that can develop later in few hours. Admission test is the short,



PATIENT UNDER GOING ADMISSION TEST ON FETAL MONITOR

continuous, electronic FHR recording made immediately on admission and gives a better impression of fetal condition. An admission test will identify those who are already at risk with an ominous pattern on admission even without contractions. In those with a normal or suspicious FHR, the functional stress of uterine contractions in early labour may bring about the abnormal FHR changes. These changes may be subtle and difficult to identify by auscultation.

For patients who are considered as low risk, a short risk, 15-20 minutes external electronic fetal monitoring on admission in labour has been suggested as screening "admission test' (Ingemarsson et al, 1986). If no fetal heart rate changes are observed with early labour consideration and the trace is normal and reactive, the chances of fetal hypoxia due to causes other than acute events are unlikely in the next few hours of labour. In a study of over 1000 low risk women conducted in Singapore, 40% of women with an ominous admission test developed fetal distress, compared to 14% in those with a reactive AT (Ingermarsson et al., 1986).

Thus admission test helps to identify a subgroup of fetuses who would benefit from more intensive monitoring while others can be monitored with intermittent auscultation.

Lawrence Tarpey, et al, (2003) carried out a randomized controlled trial on admission CTG on 8580 women admitted to delivery ward of a Dublin teaching hospital and concluded that routine use of CTG for 20 min on admission to the delivery ward does not improve neonatal outcome. No significant increase in operative delivery was apparent probably because of liberal use of fetal blood sampling.

Gary Mires, et al, (2001) also carried that randomised controlled trial of cardiotocography versus Doppler auscultation of fetal heart at admission

in labour in low risk obstetric population they concluded that compared with Doppler auscultation of fetal heart rate, admission CTG does not benefit neonatal outcome in low risk women. Its use results in increased obstetric intervention, including operative delivery.

Significance of Meconium Stained Liquor :

The presence of fresh meconium in the amniotic fluid has been 'traditionally' regarded as sign of fetal hypoxia. The passage of meconium with an increased risk of intrapartum still birth, neonatal morbidity of various degree and neonatal death. Thick meconium recognised at the onset of labour with decreased amniotic fluid volume carries the worst prognosis and is associated with a five to seven fold increased risk of perinatal death.

The first objective study associating meconium passage during labour and fetal hypoxia was done by Walker (1954) where he found that meconium passage especially thick meconium was associated with a low umbilical venous PO_2. In the presence of an abnormal FHR pattern, the passage of meconium is associated with a higher chance of the boby being aciodic and asphyxiated at birth (Miller et al., 1975; Steer et al., 1989). However, it has been shown in a number of studies that when the FHR pattern was normal, there was no difference in scalp and cord blood pH or neonatal outcome between fetuses who had or had not passed meconium (Miller et al., Shaw and Clark, 1988; Baker et al., 1992).

Meconium is present in amniotic fluid in 15% of all deliveries (Katz and Bowes, 1992). In 5% of these cases meconium is aspirated i.e. present below the vocal cords, and may lead to meconium aspiration syndrome (MAS) which is the principle cause of neonatal morbidity and mortality from the presence of meconium. Although there is no objective proof that meconium passage occurs secondary to hypoxia, it appears that meconium

aspiration is principally an intrauterine event caused by fetal hypoxia and gasping **(Katz and Bowes, 1992),** Meconium inhaled by fetus with normal acid base status and meconium inhaled at delivery are relatively begin in the absence of hypoxia (Demielium, 1994).

Sheiner E & Colleagues (2002) evaluated the effect of meconium stained amniotic fluid (AF) on perinatal outcome. A prospective observational study was performed comparing perinatal outcome of parturients with thick and thin meconium stained AF to those with clear AF. A statistically higher risk for neonatal intensive care unit admission was observed among patients with thick meconium as compared to those with clear AF. Therefore thick meconium stained AF should be considered a marker for possible fetal compromise and need careful evaluation of fetal well being.

OTHER METHODS OF INTRAPARTUM FETAL MONITORING

Fetal scalp blood ph estimation :

Fetal scalp blood sampling (FBS) for estimation of pH was introduced by Saling in 1960s, before the introduction of EFM in clinical practice.

When FHR trace is normal the chance of fetal acidosis is extremely rare (Ingemarsson, 1981), while abnormal FHR changes are not always associated with acidosis.

In the presence of continuing abnormal FHR trace, the fetal blood sampling should be repeated in 30 minutes. Sykes et al. (1982) in a study from Oxford, UK, showed that only 1 in 5 babies with 5 min Apgar <7 had severe acidosis (pH <7.1). Similarly, only 1 in 7 babies with severe acidosis had 5 minute Apgar <7, confirming that hypoxemia and acidosis are only one of the several causes of low apgar scores at birth. While the use of FBS in conjunction with continuous FHR monitoring has been shown to reduce

unnecessary operative deliveries, in certain situations the performance of FBS may lead to undue wastage of precious time. When there is an abnormal FHR pattern in early labour together with thick meconium stained amniotic fluid or when the progress of labour is unsatisfactory, caesarean section to deliver the fetus is required rather than FBS. In the presence of certain forms of pathological FHR patterns, for example prolonged, for example bradycardia (<80 beats/minute) for more than 10 minute or when presence of a sinusoidal pattern without acceleration, FBS will lead to unnecessary delay in delivery. FBS can increase the sensitivity of continuous FHR monitoring, if it is performed for a trace which on its own would not be considered to warrant immediate delivery.

FETAL SCALP STIMULATION TEST

Clark et al. (1982) were the first to observe that FHR acceleration sometimes coincided with FBS and that the scalp blood pH tended to be normal if an acceleration occurred. This lead to the development of scalp stimulation test, in which the fetal scalp is stimulated by pinching with a tissue forceps, while the CTG is observed for acceleration (Clark et al., Arulkumaran et al., 1987). If an acceleration is observed, it is highly unlikely for the scalp blood pH estimation below 7.20 and only 5% would have a scalp pH > 7.25. Absence of acceleration in non-acidnemic fetuses may be related to the use of analgesia (Spencer, 1991).

FETAL ACOUSTIC STIMULATION TEST (FAST) :

Similar to scalp stimulation test, sound stimulation with an artificial larynx placed near the region of fetal head results in an acceleration of FHR of most non-aciodotic fetuses (Smith et al., 1986). This test has a predictive value similar to the scalp stimulation test, while it is far less invasive and carries no chance of introducing any infection (Ingemarsson and Arulkumaran, 1989).

Contraction Stress Test (CST) :

The contraction stress test is one of the best available tests for the primary fetal surveillance of high risk pregnancies. The test is based on experimental evidence showing that uteroplacental blood flow decreases markedly or ceases during contraction. Therefore, uterine contractions cause a hypoxic stress that a normal healthy fetus can tolerate without difficulty. In contrast, a fetus with chronic or acute problems and will not be able to tolerate such a decrease in oxygen supply and will demonstrate this by decelerations of the FHR. Following the contractions the end point of CST is the pressure or absence of late decelerations of the FHR following uterine contractions induced by intravenous oxytocin (OCT) or by nipple stimulation.

Fetal Biophysical Profile :

Manning and colleagues (1980) proposed the combined use of five fetal biophysical variables as a more accurate means of assessing fetal health than any single variable used alone. Typically, these tests require 30 to 60 minutes of examiner time. The five biophysical variables assessed included –

1. **Fetal breathing movement :-** 30 seconds of sustained breathing movement during a 30 minute observation period
2. **Fetal movement :-** three or more gross body movements in a 30 minute observation period
3. **Fetal tone :-** one or more episodes of limb motion from a position of flexion to extension and a rapid return to flexion
4. **Fetal reactivity :-** two or more FHR accelerations associated with fetal movement of at least 15 bpm and lasting at least 15 seconds in 20 minute (reactive NST)
5. **Fluid volume :-** Presence of a pocket of amniotic fluids that measure at least 1 cm in two perpendicular planes.

Normal variables were assigned a score of two each and abnormal variables a score of zero. Thus the highest score possible for a normal fetus was 10.

Devoe LD et al. (1992) determined the ability of biophysical profile variables to predict bad perinatal outcome in high-risk third-trimester pregnancy. The outcomes of 1146 fetuses were correlated with abnormal single or multiple variables occurring in biophysical profile done within 72 hours of delivery. Two hundred forty-six fetuses had at least one abnormal biophysical profile variable with the risk of bad outcome, for a single abnormal variable, ranging from 8% (body movements) to 100% (tone) and increasing from 14% (any variable abnormal) to 63% (all variables abnormal).

They concluded that dynamic biophysical profile variables appear to be interdependent. Not all combinations of abnormal variables occur and specific combinations improve prediction of poor outcome. Risk-related scales for biophysical profile outcomes might prove superior to more conventional scoring systems.

Manning and co-authors (1993) have subsequently published a remarkable description of 493 fetuses in which biophysical scores were performed immediately before measurement of umbilical venous blood pH values obtained via cordocentesis. Approximately 20 percent of fetuses tested had growth restriction and the remaining has alloimmune anaemia. A biophysical score of zero was invariably associated with significant fetal acidemia, where as normal score (8 or more) was associated with normal pH.

Kamel HS, & colleagues (1999) followed a total of 330 high risk pregnant women with gestational ages of 32-42 weeks until delivery using the biophysical profile (BPP) and a screening test consisting of the amniotic fluid

index together with fetal acoustic stimulation under ultrasound M-mode scanning. The test was compared with the BPP and nonstress test (NST) for predicting abnormal outcome. The sensitivities of the BPP, NST and the proposed test were 100, 98.4 and 100% respectively, the negative predictive values were 100, 99.4 and 100%, while the false- positive rates were 21.3, 39.3 and 67%. Our simplified biophysical testing method is suggested to be a good negative preliminary screening test, while positive results require further fetal testing methods. This test reduced the need for BPP in many high-risk patients and had the advantages of simplicity, low cost and less time consumption.

Habek D, Hodek B & colleagues (2001) evaluated variables of the biophysical profile in the assessment of perinatal outcome. The prospective study included 87 pregnant women with singleton pregnancy in the 28th to 42nd week of gestation with clinically and ultrasonically verified fetal growth retardation. The most sensitive variable of the biophysical profile in the prediction of perinatal outcome was the amniotic fluid volume, followed by fetal breathing movements, non-stress test and fetal movements, while the lowest prediction value was assigned to the fetal tone.

Hadar A, Sheiner E & Colleagues (2001) evaluated perinatal outcomes of infants who had pathologic fetal heart rate tracing during the first stage of labour. The preinatal outcomes of 301 infants born at 37 to 42 weeks of gestation with pathologic fetal heart rate patterns during the first stage of labour were compared with 300 infants with normal fetal heart rate tracing patterns. Late decelerations and severe variable decelerations (<70 bpm) during the first stage of labour were independent risk factors (odds ratio, 17.5; 95% Cl, 1.61%-185.7% and odds ratio, 3.9; 95% Cl, 1.36%-11.7%, respectively) that were associated with fetal acidosis (determined by both pH of <7.2 and a base deficit of 12 mmol/L or higher). The operative

delivery rate was higher among patient with abnormal first stage fetal heart rate patterns.

MODIFIED BIOPHYSICAL PROFILE

It includes non-stress test and ultrasonic assessment of amniotic fluid. Measurment of amniotic fluid is an important part of antepartum fetal monitoring. Until 16 weeks of the gestationat age, the amniotic fluid is similar to that of extra cellular fluid of the fetus, no particulate matter is present and upto 16 weeks amniotic fluid is a product of amniotic membrane covering the placenta, cord and fetal membranes.

After 4 months of gestation fetus participates in modifying the volumes and composition of amniotic fluid by micturating and swallowing progressively large amount of liquor and contains more urea and .uric acid than plasma but Na-K+ are tow so the osmolality remains low.

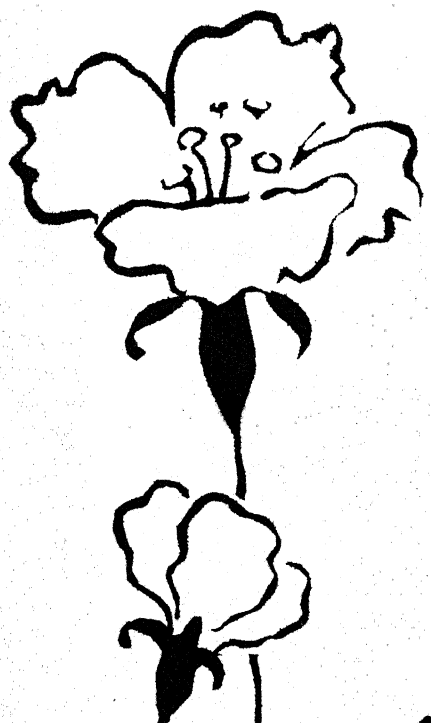
Diminished fluid volume is termed as oligohydramnios and >2000 ml is considered excessive known as polyhydramnios. Estimation of amniotic fluid is done by amniotic fluid index. This is calculated by adding the vertical depths of largest pocket in each of four quadrants by placing a linear ultrasound transducer perpendicular to the wall of uterus and parallel to the mother's spine. Pockets consisting primarily of umbilical cord are discarded. A four quadrant sum of 5 cm or greater is considered normal.

In 1980 Manning and coworkers proposed that a 'pocket' of fluid measuring less than 1 cm in vertical dimension signified pathological oligohydramnios. Crowfy and coworkers in 1984 used 4 cm pocket dimension to predict fetal distress in post term pregnancy.

In **1987 Jeffery P Phelan, Susan E Rutherford** gave the technique of four quadrant approach for estimation of amniotic fluid index. The purpose

of this investigation was to correlate non- stress test results and pregnancy outcome with amniotic fluid measurement using 4-quadrant technique in a group of high-risk pregnancy. They concluded that there is a direct relation between low amniotic fluid index and non-reactive NST, FHR deceleration, meconium staining, caesarean section for the fetal distress and low apgar scores.

Ghosh G and Colleagues (2002) gave amniotic fluid in low risk pregnancy as an admission test to the labour ward. 600 low risk pregnancies were included in study. 267 women had ruptured membranes. Among these a significant increase in operative delivery because of fetal distress was seen in cases of oligohydramnios compared with the normal amount of amniotic fluid (odds ratio 3.86, confidence interval = 1.25-11.9). The group with intact membranes comprised 333 parturient. Among these, no significant differences in perinatal outcome could be seen in relationship to the amniotic fluid index, although a 50% increase in emergency operations for fetal distress was seen in women with oligohydramnios. The results indicate that measurement of the amniotic fluid index in low risk pregnant women admitted for labour might identify parturient with an increased risk of intrapartum fetal distress.



Material and Methods

Material And Methods

A random population of pregnant females admitted to labour room in Department of Obstetrics and Gynaecology, Maharani Laxmi Bai Medical College, Jhansi were studied as cases for present study. 150 cases were taken for study. All cases were clinically evaluated by taking a detailed history and doing general, systemic as well as obstetrical examination at the time of their admission with special reference to their-

- LMP/EDD, age and parity
- Presence or absence of any high risk factor
- History of leaking etc.

Following high risk factors are noted:

- Elderly primipara
- Grand multipara
- Bad obstetric history
- Placenta previa
- Previous LSCS
- Pregnancy induced hypertension
- Post term pregnancy
- Oligohydramnios
- Previous gynaecological surgery
- Intrauterine growth retardation
- Breech pregnancy
- Rh isoimmuunization
- Any other medical illness like:
  - Anaemia
  - Hypertension

- Urinary tract infection
- Diabetes
- Heart disease
- Renal disease etc.

- Investigations were noted
- Based on the presence or absence of high risk factor the patients were divided in two groups:
  - High Risk Group (99) patients
  - Low Risk (51) patients
- In all the patients included in the study an abdominal ultrasound including amniotic fluid index was done.

In all patients included in the study a non-stress test (admission test) was done at the time of admission.

Patients were monitored for 20 minutes in a semilateral position on fetal monitor. The record of detailed findings of non-stress test were noted down. All these patients were watched during labour in a routine fashion. Findings of non-stress test were kept blinded with the labour room team. Perinatal outcome was noted in all the patients in form of:

- Duration of labour
- Fetal weight/sex
- Apgar Score
- Mode of delivery
  - vaginal
  - Abdominal
- Duration of labour
- Meconium stained liquor
- Need for endotracheal intubation of baby

- Need for admission to NNU
- Duration of hospital stay of baby
- Neonatal hospital nursery deaths

All the data was compiled and analysed and conclusion were drawn regarding the correlation between findings of admission test and fetal outcome parameters:

During the interpretation of admission test, a systematic approach was used to assess four aspects of fetal heart rate tracing and according to them; non-stress test was classified in three groups:

1. Normal/reassuring/Reactive

- Baseline heart rate 110-150 bpm
- Baseline variability 10-25 bpm
- Fetal movements at least two
- Presence of accelerations of at least > 15 bpm lasting > 15 sec.
- Absence of decelerations

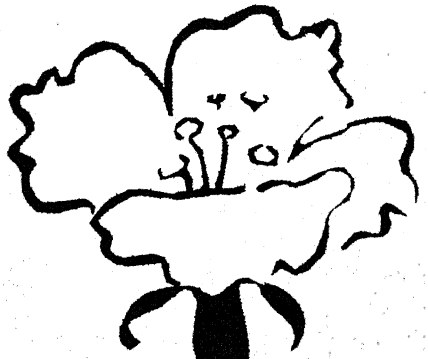
2. Suspicious/Equivocal

- Baseline heart rate 150-170 bpm or 100-110 bpm
- Reduced baseline variability 5-10 bpm for > 20 min with normal baseline and no deceleration
- Fetal movements < 2
- Absence of accelerations for > 40
- Variable decelerations <60 bpm for <60 sec.

3. Pathological/Omnious/Non reactive

- Baseline heart rate <100 bpm or > 170 bpm
- Silent pattern <5 bpm variability >40 min
- Fetal movements – absent

- Deceleration variable >60 bpm lasting for >60 sec or repetitive late decelerations
- Sinusoidal pattern (oscillation frequency < 2-5 cycles/min amplitude of >10 bpm) with no acceleration.



Observations

Observations

Present study entitled, "Role of admission test as a screening procedure for perinatal outcome" was carried out on patients admitted in the labour room of Department of Obstetrics and Gynaecology, Maharani Laxmi Bai Medical College, Jhansi from September 2003 to August 2004. A total number of 150 patients were selected randomly. Cases included in the present study were both from high risk as well as low risk group admitted in early stage of labour. These patients were subjected to undergo a non-stress test on admission and result was concealed from labour room team. All these patients were watched during labour in a routine fashion.

Data obtained after the study is given in following tables:

Table I

RESULTS OF ADMISSION TEST IN TOTAL PATIENTS

(n = 150)

Results of admission test	Number of cases	Percentage (%)
Reactive	106	70.67
Equivocal	28	18.67
Non-reactive	16	10.67
	n = 150	100%

Table I shows result of non-stress test among the study group. Out of 150 patients included in the study 106 (70.67%) reactive tracing, 28 patients (18.67%) showed equivocal tracing and 16 patients 10.67% showed non-reactive tracing on admission (shown in figure 1).

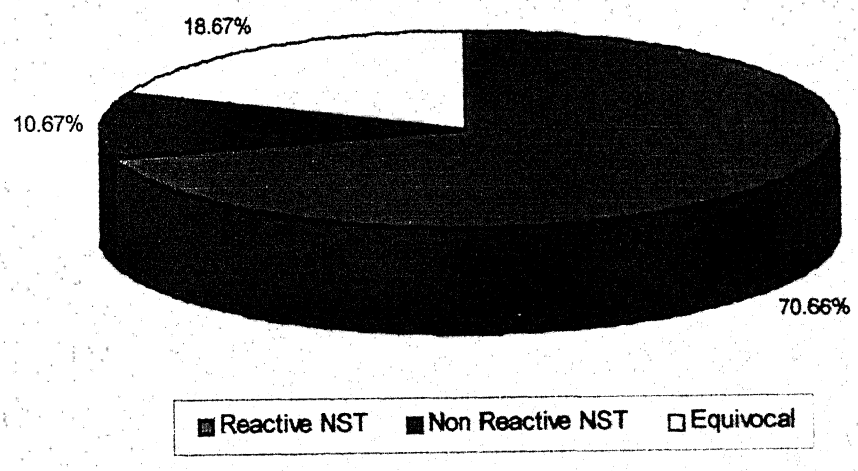


Fig. 1 : RESULTS OF ADMISSION TEST IN STUDY GROUP (n=150)

Table II

DISTRIBUTION OF RESULT OF ADMISSION TEST AMONG PRIMIGRAVIDA PATIENTS ($G_1 P_{0+0}$) (n = 72)

Results of Admission test	Number of cases	Percentage (%)
1. Reactive NST	49	68.05
2. Abnormal NST Equivocal Non Reactive	14 | 23 9	31.94
	72	100

Table II shows distribution of admission test result among primigravidas ($G_1 P_{0+0}$). Out of 72 primigravidas in the present study 49 (68.05%) showed reactive tracing while 23 patients (31.94%) showed abnormal non-stress test results.

The difference when calculated statistically was found to be insignificant ($p>0.05$).

Table III

DISTRIBUTION OF BASELINE HEART RATE IN TOTAL PATIENTS (n = 175)

Baseline Heart Rate (in Beats/min.)	Number of cases (n)	%
< 100	0	0
101-110	3	2
111-150	120	80
151-170	26	17.33
> 170	1	0.67
	n = 150	100

Table III shows baseline heart rate among the study group. Out of 150 patients, 120 (80%) showed baseline heart rate in the range of 110-150 beats/min. 17.33% patients showed borderline tachycardia.

Table IV

ASSOCIATION BETWEEN RESULT OF ADMISSION TEST AND GESTATIONAL AGE

Result of admission test	Number of cases in group A (<37 weeks)		Number of cases in group B (37-40 weeks)		Number of cases in group C (≥ 40 weeks)	
	No.	%	No.	%	No.	%
1. Reactive	12	80	73	73	21	60
2.Abnormal NST	3	20	27	27	14	40
Total (n=150)	n= 15		n = 100		n = 35	

Table IV shows distribution of result of admission test among different gestational age group patients. Out of 100 patients in gestational age group 37-40 week, 73 patients (73%) showed reactive admission test while 27 patients (27%) showed abnormal non-stress test. While out of 15 patients in group with gestational age <37 weeks, 12 patients (80%) showed reactive tracing and 3 patients (20%) showed abnormal tracing. In group with gestational age ≥ 40 weeks, 21 patients (60%) showed reactive non-stress test while 14 patients (40%) showed abnormal results. .

When the difference of results was compared in both A (<37 week) and C (>40 weeks) groups with group B (37-40 weeks), no significant relation ($p>0.05$) could be established except that it was seen that there was higher risk of NST being abnormal when gestational age was more than >40 weeks.

Table V

RELATION OF RESULT OF ADMISSION TEST WITH RISK FACTOR

Result of admission test	Cases with risk factor		Cases without risk factor	
	No.	%	No.	%
1. Reactive	64	64.64	42	82.36
2. Abnormal NST	35	35.35	9	17.64
Total (n= 150)	N = 99		N = 51	

Table V shows relation of result of admission test with risk factors. Out of 150 patients, 99 patients (66%)showed one or more high risk factors. The patients who were having high risk factors, 64 patients (64.64%) had reactive non stress test 35 patients (35.35%) had abnormal non stress test.

Among 51 patients without any high risk factor 42 patients (82.36%) showed reactive admission test, while 9 patients (17.64%) still showed abnormal non stress test.

It was seen that there was high risk of NST being abnormal in high risk patients. When these two groups were compared statistically, this difference was found to be significant ($p<0.05$).

Table VI

RELATIONSHIP OF ADMISSION TEST WITH INDIVIDUAL RISK FACTOR (N=99)

High Risk Factor	No. of Cases	Reactive Admission Test			Abnormal Admission Test		
		No.		%	No.		%
Pregnancy induced hypertension PIH Alone PIH with IUGR PIH with other risk factors	n =19 8 3 8 (19.19%)	7 2 5	14	73.68	1 1 3	5	26.31
Intrauterine growth retardation IUGR Alone IUGR with oligohydramnios	n =14 8 6 (14.14%)	6 2	8	57.14	2 4	6	42.85
3. Postdatism	n=35 (35.35%)	21		60	14		40
4. Decreased fetal movements	n=14 (14.14%)	9		64.28	5		35.71
5. Anaemia	n=6 (6.06%)	4		66.67	2		33.33
6. Previous Abortion	n=20 (20.20%)	14		70	6		30
7. Previous still birth	n=8 (8.08%)	6		75	2		25
8. Heart disease	n=4 (4.04%)	3		75	1		25
9. Previous LSCS	n=19 (19.19%)	12		63.15	7		36.84
10. Rh-ve	n=4 (4.04%)	3		75	1		25
11. Polyhdramnios	n=1 (1.01%)	1		100	-		-
12. Others	n=10 (10.10%)	7		70	3		30

Table VI shows distribution of different risk factors among the study groups. Most commonly encountered risk factor was postdatism (i.e. gestational age > 40 weeks). Out of 99 patients, 35 patients (35.35%) were postdated. Other important risk factor was pregnancy induced hypertension which was found in important risk factor was pregnancy induced hypertension which was found in 19 patients (19.19%). Intrauterine growth retardation was found in 14 patients (14.14%). Out of these intrauterine growth retardation associated with severe oligohydramnios was present in 6 patients (i.e.-42.8%) 14 patients (14.14%) had decreased fetal movements.

In case of pregnancy induced hypertension 2.31% patients; in patients with intrauterine growth retardation 42.85% patients, in postdated patients 40% patients and in case of decreased fetal movements 35.71% patients showed abnormal non stress test results.

Table VII

RELATIONSHIP OF RESULTS OF ADMISSION TEST WITH POSTDATED PREGNANCY (n = 35)

Result of Admission test	Cases with Postdated Pregnancy	
	No.	%
1. Reactive	21	60
2. Abnormal NST	14	40
	n = 35	

Table VII shows relationship of admission test result with postdated pregnancy. Out of 35 patients with postdated pregnancy 21 patients showed reactive tracing where as 14 patients (40%) showed abnormal non stress test result.

Although there was relative risk of NST being abnormal in postdated pregnancy but no significant relation could be established when statistically calculated ($p>0.05$).

Table VIII

RELATIONSHIP OF RESULTS OF ADMISSION TEST WITH PREGNANCY INDUCED HYPERTENSION (n = 19)

Result of Admission test	Cases with Pregnancy Induced Hypertension	
	No.	%
1. Reactive	14	73.68
2. Abnormal NST	5	26.31
	n = 19	

Table VIII shows association of admission test result with pregnancy induced hypertension. Out of 19 patients with pregnancy induced hypertension 14 patients (73.68%) showed reactive admission test while 5 patients (26.31%) showed abnormal admission test.

When the difference of result was calculated among cases with pregnancy induced hypertension statistically by test of proportions relation could be established ($p<0.05$).

Table IX

ASSOCIATION OF INTRAUTERINE GROWTH RETARDATION WITH RESULTS OF ADMISSION TEST (n = 14)

Result of Admission test	Cases with Intrauterine Growth Retardation	
	No.	%
1. Reactive	8	57.14
2. Abnormal NST	6	42.85
	n = 14	

Table IX shows relation of intrauterine growth retardation with result of admission test. Out of 14 patients with intrauterine growth retardation 8

patients (57.14%) had reactive admission test whereas 6 patients (42.85%) showed abnormal admission test.

There was noticeably higher risk of abnormal non stress test in patients but when statistically calculated this difference was found to be insignificant (p>0.05).

Table X

ASSOCIATION BETWEEN RESULTS OF ADMISSION TEST AND DECREASED FETAL MOVEMENTS (n = 14)

Result of Admission test	Cases with Decreased Fetal Movements	
	No.	%
1. Reactive	9	64.28
2. Abnormal NST	5	35.71
	n = 14	

Table X shows relation of decreased fetal movement perceived by patients with result of admission test. Out of 14 patients who perceived decreased fetal movements, 9 patients (64.28%) showed reactive non stress test and 5 patients (35.71%) showed abnormal admission test.

There was higher risk of abnormal admission test in patients with decreased fetal movements but statistically calculated this difference was insignificant (p>0.05).

Table XI

ASSOCIATION OF RESULTS OF ADMISSION TEST AND AMNIOTIC FLUID INDEX VALUES (Total cases = 150)

Result of Admission test	Number of cases in group A (AFI < 5 cm)		Number of cases in group B (AFI = 5-10 cm)		Number of cases in group C (AFI ≥ 10 cm)	
	No.	%	No.	%	No.	%
1. Reactive	1	25.0	48	61.5	56	82.3
2. Abnormal NST	3	75.0	30	38.4	12	17.6
Total (n=175)	n = 4		n = 78		n = 68	

Table XI shows association of result of admission test and amniotic fluid index values. Out of 150 patients in study group, 4 patients (2.6%) showed AFI value <5 cm. Among these patients,1 patient (25%) showed reactive non stress test while 3 patients (75%) showed abnormal admission test. 78 patients (52%) had AFI values 5-10 cm. Of these 48 patients (61.53%) had reactive tracing and 30 patients (38.46%) showed abnormal admission test. Rest 68 patients (45.33%) had AFI cm value >10. among these 56 (82.35%) showed reactive admission, while 12 patients (17.64%) showed abnormal admission test.

When group A was compared with both groups B & C statistically, significant differences was found. There was more chances of admission test being abnormal when AFI was <10 cm than when AFI >10 cm ($p<0.01$)

Table XII

ASSOCIATION OF RESULT OF ADMISSION TEST WITH CLINICALLY DECTECTED FETAL DISTRESS (ON AUSCULTATION) DURING LABOUR

Result of Admission test	n	Cases who developed fetal distress on . auscultation		Cases in which no fetal distress was detected	
		No.	%	No.	%
1. Reactive	106	28	26.4	78	73.5
2. Abnormal NST	44	22	50.0	22	50
	n = 150	n = 50		n = 100	

Table XII shows relation of result of admission test with clinically detected fetal distress on auscultation. Out of 106 patients who had reactive admission test, only 28 patients (26.4%) developed fetal distress, while out of 44 patients with abnormal admission test 22, patients (50.0%) developed fetal distress during labour.

When relative risk was calculated it was seen there was greater risk of development of fetal distress when NST was abnormal ($p<0.01$), sensitivity 44%, PPV 50%, specificity 78%, NPV 73.5%.

Table XIII

TIME INTERVAL BETWEEN ADMISSION TEST AND DEVELOPMENT OF FETAL DISTRESS

Result of Admission test	n	Cases developing fetal distress with 6 hr		Cases developing fetal distress in >6 hr	
		No.	%	No.	%
1. Reactive	28	13	46.5	15	53.5
2. Abnormal NST	22	13	59.1	9	40.9
	n = 50	N = 26		n = 24	

Table XIII shows time interval between admission test and development of fetal distress. Out of 28 patients with reactive admission test who developed fetal distress 13 patients (46.5%) developed fetal distress within 6 hrs whereas out of 22 patients with abnormal admission test, who developed fetal distress 13 patients (59.1%) developed distress in 6 hrs interval. It appeared that patients with abnormal NST developed fetal distress earlier than patients with reacting non stress test.

When statistically compared this difference was found to be insignificant.

Table XIV

ASSOCIATION OF RESULT OF ADMISSION TEST AND PRESENCE OF MECONIUM

Result of Admission test	n	Cases with presence of Meconium		Cases in which no meconium could be detected	
		No.	%	No.	%
1. Reactive	106	8	7.54	98	92.45
2. Abnormal NST	44	17	38.63	27	61.36
	n = 150	N = 25		n = 125	

Table XIV shows association of result of admission test with appearance of meconium during labour. Out of 106 patients with reactive non stress test only 8 patients (7.54%) showed presence of meconium, while among 44 patients with abnormal non stress test, 17 patients (38.63%) showed appearance of meconium during labour.

When relative risk was calculated it was seen there was 5.12 times greater risk of passage of meconium in patients with abnormal NST. When statistically calculated this difference was found highly significant ($p<0.001$. PPV, 38.63%, sensitivity 68%, specificity 78.4%, NPV 92.45%).

Table XV

MODE OF DELIVERY IN DIFFERENT GROUPS

Result of Admission test	N	Cases delivered abdominally by LSCS		Cases delivered vaginally	
		No.	%	No.	%
1. Reactive	106	44	41.5	62	58.4
2. Abdominal NST	44	25	56.8	19	43.2
	n = 150	n = 69		n = 81	

Table XV shows association of result of admission test with mode of delivery. Out of 106 patients with reactive non stress test, 44 patients (41.5%) delivered by abdominal route by LCS and 62 patients (58.4%) delivered by vaginal route. Out of 44 patients with abnormal admission test 25 patients (56.8%) had LSCS done while 19 patients (43.2%) delivered vaginally.

Although it was seen that there was greater risk of abdominal delivery in case of abnormal non stress test but this association was not found to be significant when significantly evaluated ($p>0.05$).

Table XVI

ASSOCIATION OF RESULT OF ADMISSION TEST WITH APGAR SCORE OF BABY AT 5 MINUTES

(TOTAL CASE – 150)

Result of Admission test	n	Cases in which APGAR was (7-10)		Cases in which APGAR <7	
		No.	%	No.	%
1. Reactive	106	102	96.2	4	3.7
2. Abdominal NST	44	36	81.8	8	18.1
	150	138		12	

Table XVI shows association of result of admission test with APGAR score at 5 min. Among 106 patients with reactive tracing babies of 102 patients (96.2%) had APGAR 7-10, while only 4 babies (3.7%) showed APGAR score less than 7. Contrary to this in patients with abnormal admission test result, babies of 36 patients (81.8%) showed APGAR 7-10 and 8 babies (18.1%) had APGAR score <7 at 5 min.

When relative risk was calculated it was seen that there was 4.89times more risk of perinatal asphyxia in patients with abnormal admission test. This association when statistically calculated was found to be highly significant ($p<0.01$, PPV 18.8% sensitivity 66.67% specificity 73.91%, NPV 96.22%).

Table XVII

ASSOCIATION OF RESULT OF ADMISSION TEST WITH ADMISSION OF BABIES TO NEONATOLOGY UNIT (TOTAL NO. OF CASES = 150)

Result of Admission test	n	Number of Babies Admitted to Neonatology Unit		Number of Babies in which there was no admission to NNU	
		No.	%	No.	%
1. Reactive	106	7	6.6	99	93.4
2. Abdominal NST	44	14	31.8	30	68.1
	150	21		129	

Table XVII shows association of result of admission test with admission of babies to neonatology unit. Out of 106 patients who showed reactive admission test, babies of 7 patients (6.6%) were admitted to neonatology units. Whereas out of 44 patients with abnormal non stress test tracings, babies of 14 patients (31.8%) were admitted to neonatal unit because of perinatal asphyxia.

When relative risk was calculated it was found that the there was 4.81 times more risk of admission to neonatology unit when admission test was abnormal and this association was found to be highly significant ($p<0.001$, PPV 31.8%, sensitivity 66.67%, specificity 76.74%, NPV 93.39%).

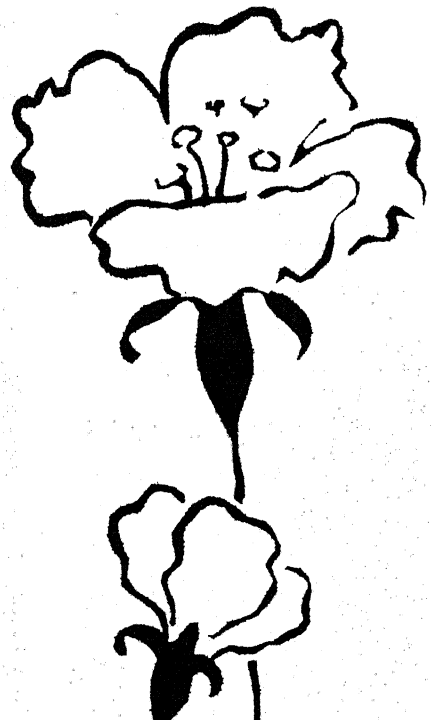
Table XVIII

ASSOCIATION OF RESULT OF ADMISSION TEST WITH PERINATAL MORTALITY

Result of Admission Test	n	Babies expired in perinatal period	
		No.	%
1. Reactive	106	2	1.8
2. Abnormal NST	44	4	9.09
		N = 6	

Table XVIII shows association of result of admission test with perinatal mortality in the study group. Out of 106 patients, who had reactive tracing 2 babies (1.8%) expired in neonatal period. Whereas out of 9.09% patients with abnormal admission test, babies of 4 patients (9.09%) expired in early neonatal period.

There was found to be 5.05 times more risk of perinatal mortality in patients with abnormal admission test when statistically calculated this difference was found to be insignificant.



Discussion

Discussion

Present study ***"Role of Admission test as a Screening procedure for perinatal Outcome"*** was carried out to assess the importance of admission test in detecting fetal asphyxia in utero at the time of admission in early labour and correlating it with fetal parameters at the time of birth.

The cases included in the study were selected randomly from those admitted in early labour after 34 weeks of gestation. They consisted of patients with high risk factor as well as without high risk factor.

Studies in past recognised that the results of intrapartum fetal heart rate monitoring correlate with immediate fetal outcome (Hausmacher *et al.*, 1968; Beard *et al.*, 1971; Fox *et al,* 1976; Ingemarsson, S. Arulkumaran, 1986). Other studies have questioned whether similar association can be drawn between antenatal fetal cardiotocography and fetal outcome (Visser and Huisjes. 1977;Liu et al. 1978).

After doing a non-stress test at the time of admission the patients were followed in routine manner by ausultation as well as other important factors were noted. In some patients oxytocin acceleration was done.

A total of 150 patients were taken for study. There were 72 primigravidas and 78 were multigravidas, 3 patients were teenagers and 3 patients were elderly gravidas. No statistical association was found between parity of patients and the result of admission test ($p>0.05$).

It is established that with increasing fetal maturation the mean heart rate decreases (Pillai and James, 1990). It is postulated that this normal gradual slowing of fetal heart rate corresponds to maturation of parasympathetic (vagal) heart control (Renou and coworkers, 1969). During

third trimester, the normal average baseline heart rate is 110-150 beats/minute (FIGO Guidelines, 1987). During present study 80% patients had their baseline heart rate in the range of 110 - 150 beats/minute. 26 patients (17.33%) showed borderline tachycardia *i.e.* baseline heart rate between 150-170 beats/minute (shown in Fig. 2). Out of these patients 40% patients developed fetal distress later during the labour. It was also seen that there was greater incidence of fetal morbidity and mortality when tachycardia was associated with one or more associated abnormality on non-stress test.

Pilial and James (1990) reported that upto 30 weeks, baseline characteristics were similar during both fetal rest and activity. About 40% of postdated patients show non-reassuring fetal heart rate patterns (Fernado Arias, Hannah colleagues, 1992). During the present study although 80% patients of gestational age <37 weeks showed reactive non stress test, no definite association could be drawn between these and the term patients ($p>0.05$). When the results were compared between group with gestational age 37-40 weeks and postdated patients although it was seen that there were greater chances of NST being abnormal in later group but yet no significant association could be established ($p>0.05$) (shown in Fig. 3).

Out of 150 patients 99 women carried one or more high risk factors. Risk of having abnormal NST was more in patients with high risk factor (RR 1.82). Different high risk factors were postdatism, pregnancy induced hypertension, intrauterine growth retardation, decreased fetal movement, anaemia, Rh-isoimmunization, oligohydramnios *etc.* (shown in Fig. 4).

In pregnancy induced hypertension, 26.31% patients showed abnormal non-stress test. Among the patients with abnormal NST, 75% delivered by

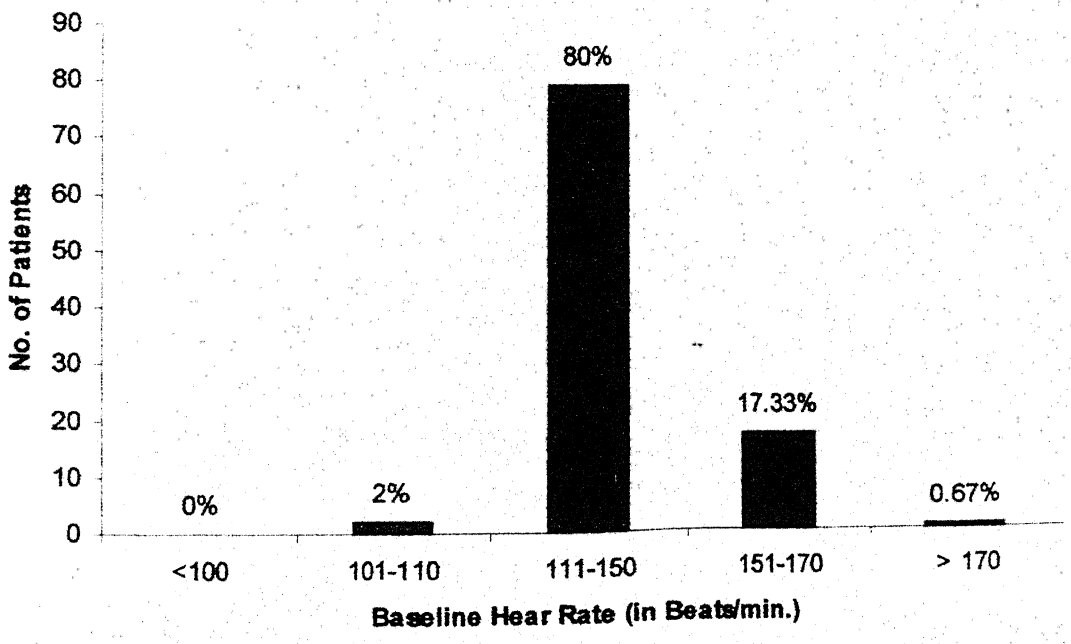


Fig. 2 : BASELINE HEAR RATE IN STUDY GROUP (n=150)

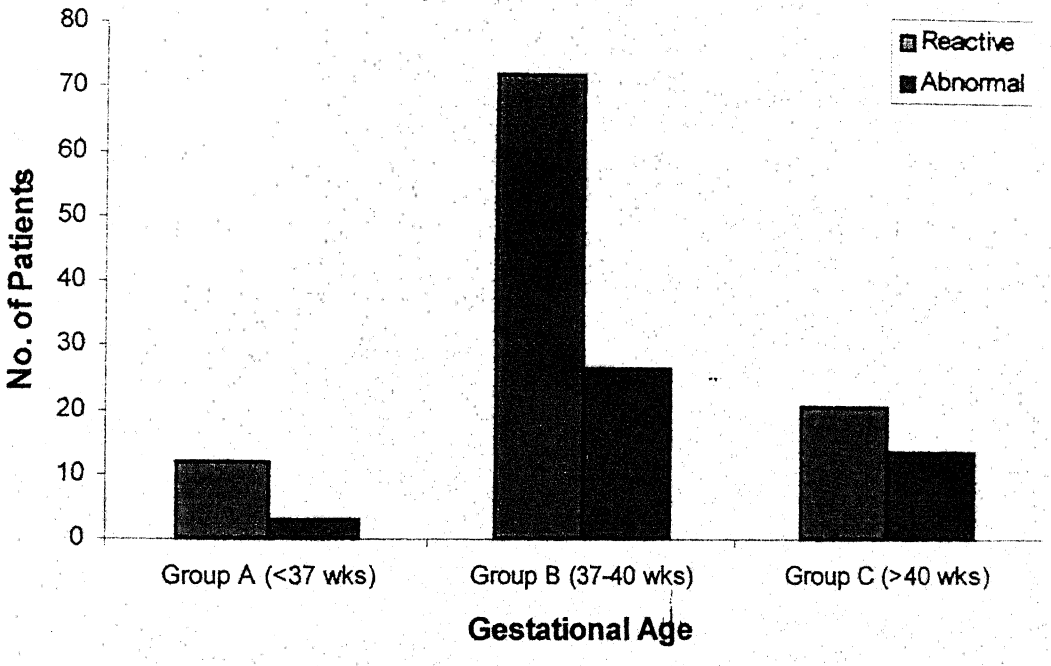


Fig. 3 : ADMISSION TEST RESULTS IN PATIENTS OF DIFFERENT GESTATIONAL AGE GROUPS

lower segment cesarean section. 2 babies were admitted in NNU because of perinatal asphyxia & 1 of them expired after 3 days.

42.85% patients with intrauterine growth retardation showed abnormal admission test. More than 60% patients showed this type of pattern when intrauterine growth retardation was associated with oligohydramnios. Babies of 5 patients with intrauterine growth retardation expired in neonatal period. Out of these one baby had fatal congenital anomalies and most of them died due to meconium aspiration syndrome or RDS. One baby was kept on oxygen and intravenous antibiotics and was discharged after 10 days.

Among postdated pregnancies, 40% patients showed abnormal non-stress test, of which 62% patients had abdominal delivery. 3 patients were post term (>42 weeks). One of them showed non-reactive NST and had lower segment cesarean section for fetal distress. Baby was admitted in neonatology unit for 10 days

Other high risk factor encountered was decreased fetal movements. Out of 14 patients, 35.71% patients showed abnormal admission test. 75% patients had cesarean section done for fetal distress. Babies of 2 of these of were for meconium aspiration admitted in NNU.

During the study 6 patients of severe anaemia were encountered. One of these patients had IUGR along with anaemia and showed non-reactive non- stress test. Patient had vaginal delivery and baby was admitted to NNU because of meconium aspiration.

In the present study, amniotic fluid index was done in all the cases by four quadrant method. Earlier, role of AFI in prolonged pregnancy was explained by Crowley (1980) and Leveno *et al.* (1984). They showed that fetal compromise in prolonged pregnancy was due to cord compression due

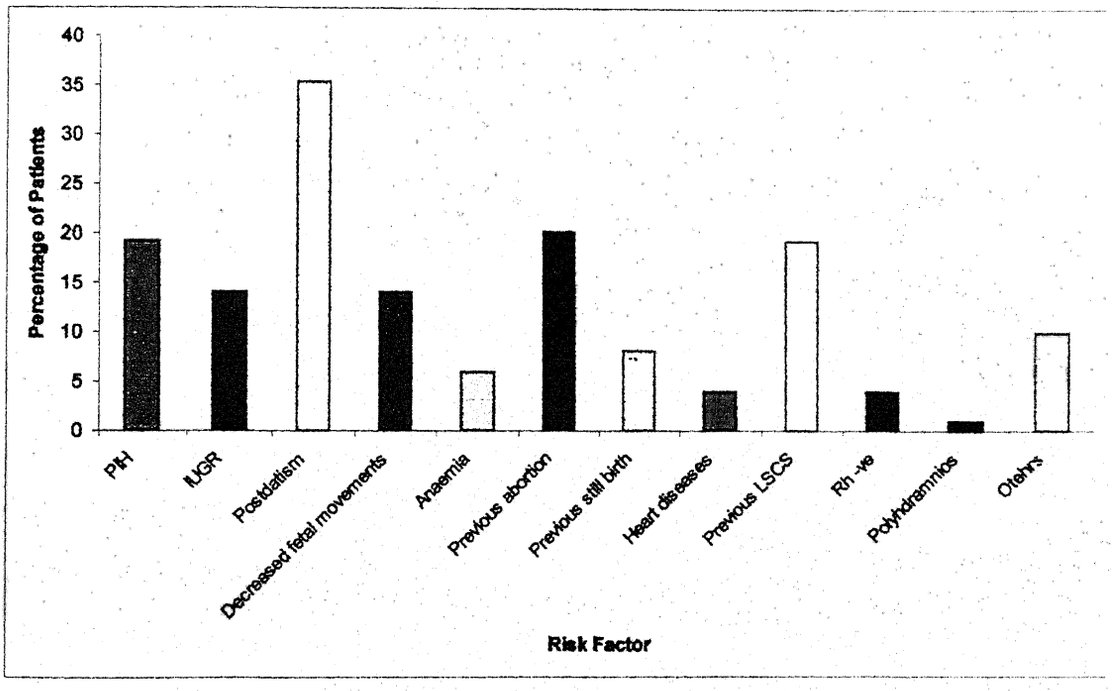


Fig. 4 : DISTRIBUTION OF DIFFERENT HIGH RISK FACTORS IN STUDY GROUP

to low AFI (Oligohydramnios). 4 patients in the study group had oligohydromnios *i.e.* AFI <5 cm. 75% patients in this group had abnormal NST and all 4 patients developed fetal distress (shown in Figure 5). 3 patients had LSCS and one patient had vaginal delivery. In this group 50% babies had perinatal mortality. One patient had polyhydramnios (*i.e.* AFI >20 cm). Admission test showed reactive result but patient had LSCS for cord presentation. It was seen from the study that there were greater cases showing abnormal non-stress test when AFI was <10 cm when compared with cases with AFI >10 cm ($p<0.05$).

Ingemarsson *et al.* (1986) also carried out a study in low risk patients. Study was carried out in over 1000 low risk women and it was seen that 40% of patients with omnious admission test developed fetal distress compared to 1.4% with reactive admission test. While in present study there were 51 patients from low risk group. Of these patients 42 (82.37%) patients showed reactive nonstress test and 9 (17.64%) showed abnormal NST. In the abnormal NST group 22.2% patients developed fetal distress compared to reactive NST group in which 19.04% patients developed fetal distress. When incidence of fetal distress was compared in high and low risk groups difference was found significant (p-<0.01). But no definite association could be established between risk factor and development of fetal distress in abnormal NST group ($p>0.05$) (shown in Figure 6).

In present study it was seen that there was 1.8 times more risk of developing fetal distress in patients with suspicious/non-reactive non stress test ($p<0.01$; specificity 78%, NPV 73.5%) when compared with cases with reactive NST. It was seen during the follow up that patients with abnormal NST developed fetal distress earlier as compared to the patients with reactive NST but no statistical significance could be established.

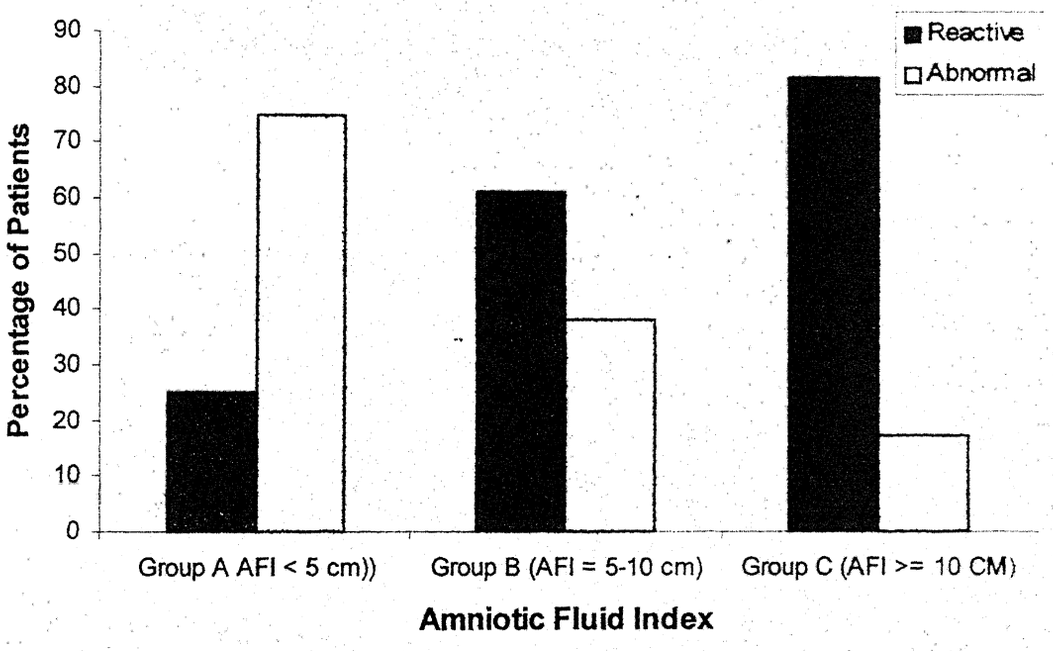


Fig. 5 : CORRELATION OF RESULTS OF ADMISSION TEST AND AMNIOTIC FLUID INDEX VALUE IN STUDY GROUP

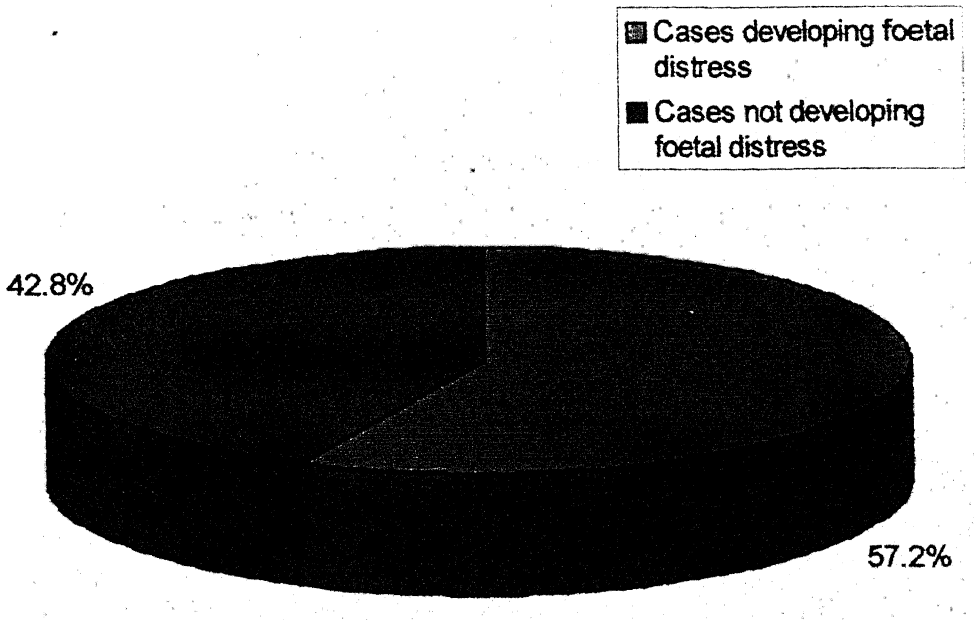


Fig. 6a : CASES WITH ABNORMAL NST IN HIGH RISK GROUP (n=35)

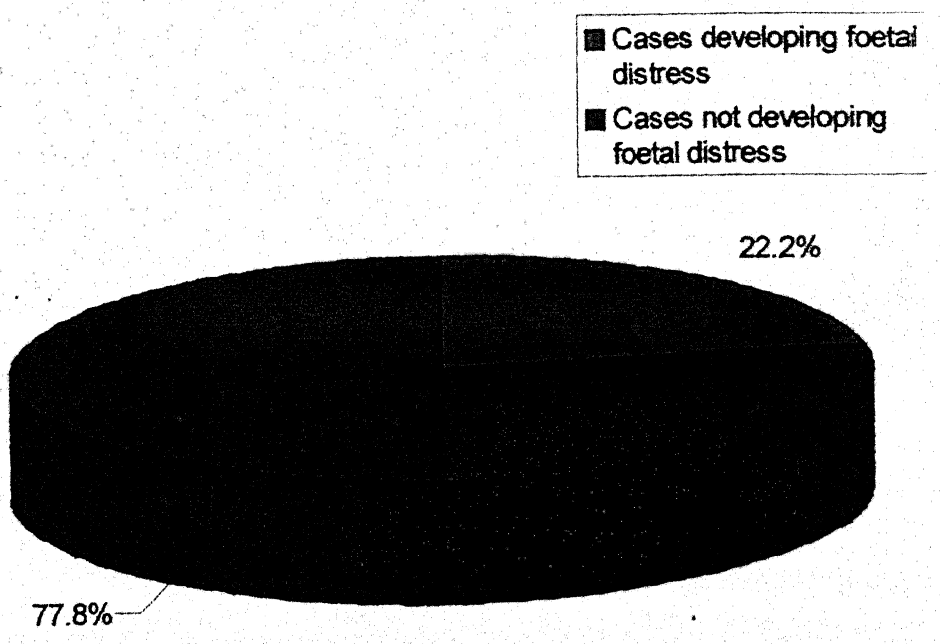


Fig. 6b : CASES WITH ABNORMAL NST IN LOW RISK GROUP (n=9)

Presence of fresh meconium in amniotic fluid has been traditionally regarded as a sign of fetal hypoxia. Several studies have been carried out to show that passage of meconium is associated with higher risk of intrapartuin still birth, neonatal morbidity of various degrees and neonatal death (Miller *et al.*,1975, Steer *et al.*, 1989). During present study 25 patients showed passage of meconium, of which 68% patients were from suspicious/non-reactive group (shown in Figure 7), when this was evaluated statistically it was found to be highly significant ($p<.0001$, specificity 78.4%, NPV 92.45, Senstivity 68%). It was seen that there was 5.12 times higher risk of passage of meconium in patients with abnormal admission test. Out of 25 patients with meconium 71.4% patients developed fetal distress clinically in labour later on. In patients who had reactive NST and passed meconium during labour 75% patients delivered vaginally and 2 babies showed meconium aspiration syndrome. One baby expired because of fatal congenital anomalies whereas in patients with abnormal admission test there was higher rate of cesarean deliveries because of fetal distress. There were 6 neonatal admissions and out of these 2 babies showed neonatal death. Rest of the babies were discharged with in 7-10 days. This showed that in patients with abnormal NST as well as passage of meconium, the fetal morbidity and mortality was higher.

It was seen that in institutions where continuous electronic monitoring was done the rate of cesarean sections has increased without any decline in perinatal mortality rates (Klein *et* al. 1993; MacDonald, 1985). During the present study out of 44 patients with abnormal test 25 patients (56.81%) were delivered abdominally as compared to 19 (43.18%) who were delivered vaginally (Shown in figure 8). Statistically this difference was found to be insignificant.

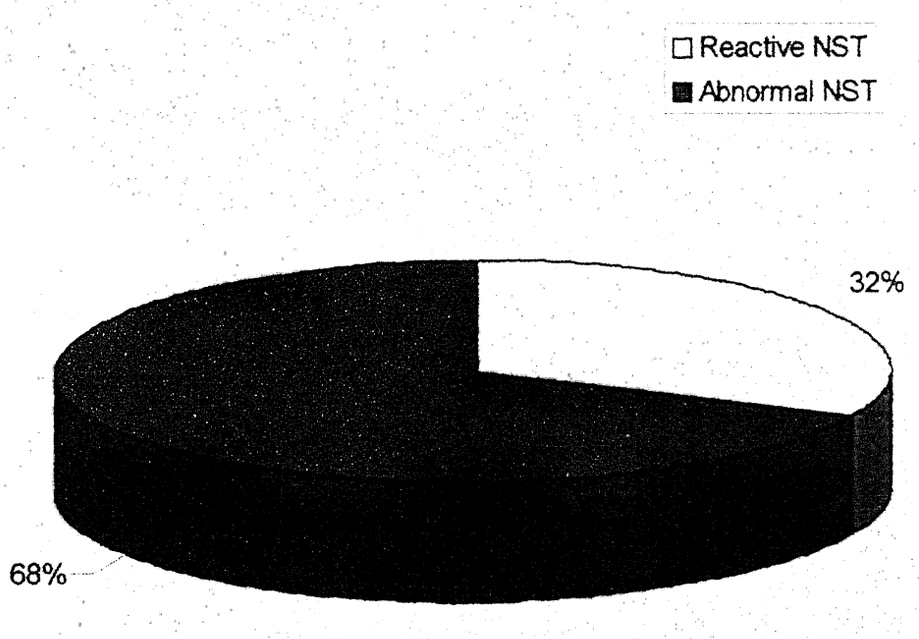


Fig. 7 : CASES WITH PRESENCE OF MECONIUM (n=25)

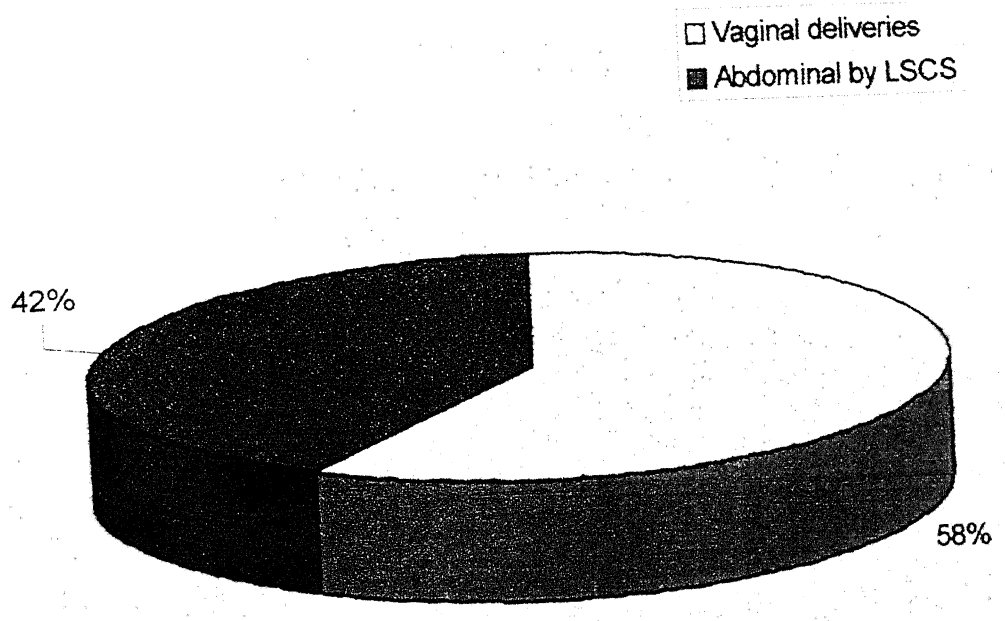


Fig. 8a : MODE OF DELIVERY IN REACTIVE NST GROUP (n=106)

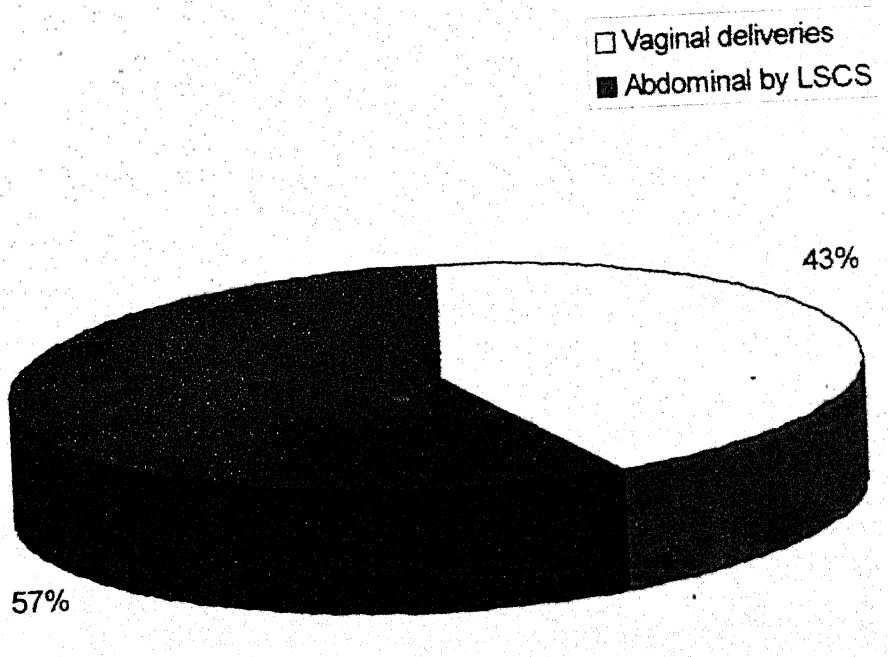


Fig. 8b : MODE OF DELIVERY IN ABNORMAL NST GROUP (n=44)

In a study carried out by Beard *el al.* (1971), Steer *el al.* (1989) showed that in presence of a normal FHR trace there was only 2% chance of fetus to be acidotic (pH <7.2) and a 1% chance for it to have a 5 minute Apgar score of <7. Some other studies showed that hypoxia and acidosis are unlikely in presence of accelerations and normal baseline variability (Ingemarsson and Arulkumaran, 1989; Arulkumaran *et al.,* 1987: Ingemarsson *et al.,* 1993) and there are more babies with low apgar scores and those who need admission to the neonatal intensive care unit for asphyxia in patients with abnormal NST (Dunphy et al. 1991).

During present study in patients with reactive non-stress test only 3.77% patients showed apgar score <7 whereas in case of abnormal admission test, 18.18% patients showed apgar <7 at 5 minute (shown in Figure 9). This association of abnormal NST and lower apgar score of baby at 5 minute was found to be statistically significant. There was 4.82 times higher risk of low apgar score in cases with abnormal NST at admission (sensitivity 66.67% specificity 73.91% NPV 96.22%).

When neonatal admissions were compared in both groups of admission test, it was seen that there were 4.81 times more neonatal admissions in patients with abnormal admission test (p-<.001, Specificity 76.74%, NPV 93.39% Sensitivity 66.67%) (shown in Figure 10).

During the present study it was seen that perinatal mortality was more (9.09%) in cases with abnormal NST when compared with reactive NST (1.8%). Relative risk was 5.05 times higher in abnormal test group but when statistically calculated the difference was found to be insignificant.

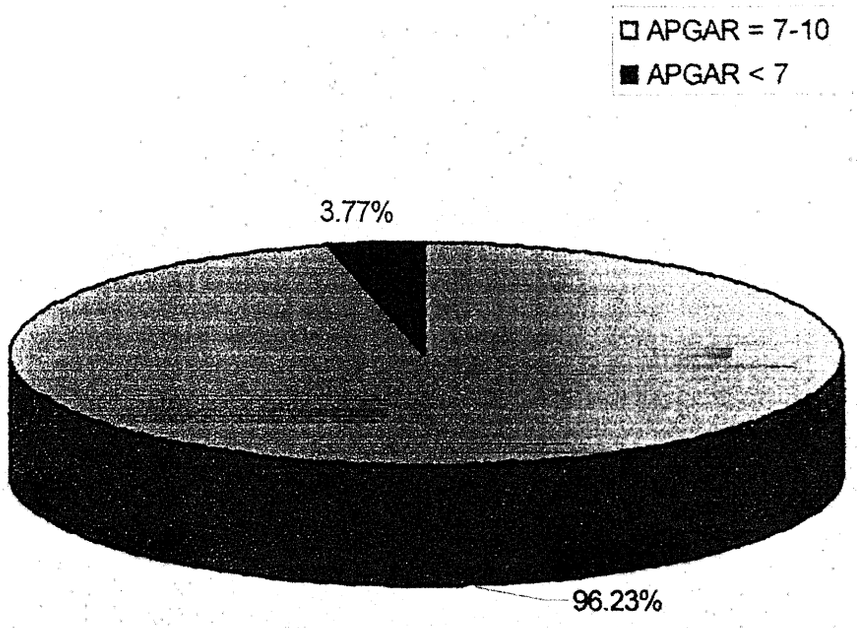


Fig. 9a : APGAR SCORE OF BABIES AT 5 MIN. IN REACTIVE NST GROUP (n=106)

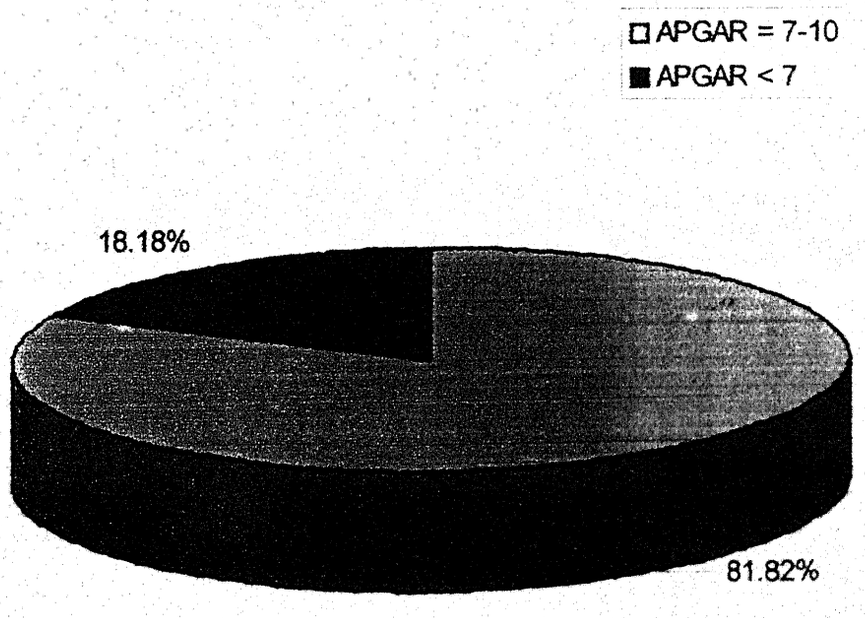


Fig. 9b : APGAR SCORE OF BABIES AT 5 MIN. IN ABNORMAL NST GROUP (n=44)

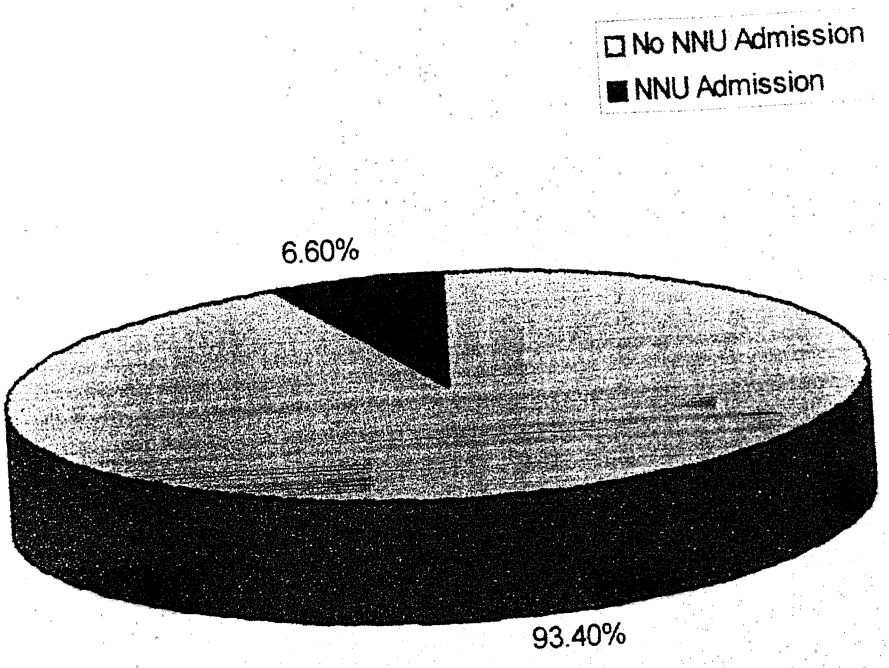


Fig. 10a : NNU ADMISSION IN REACTIVE NST GROUP (n=106)

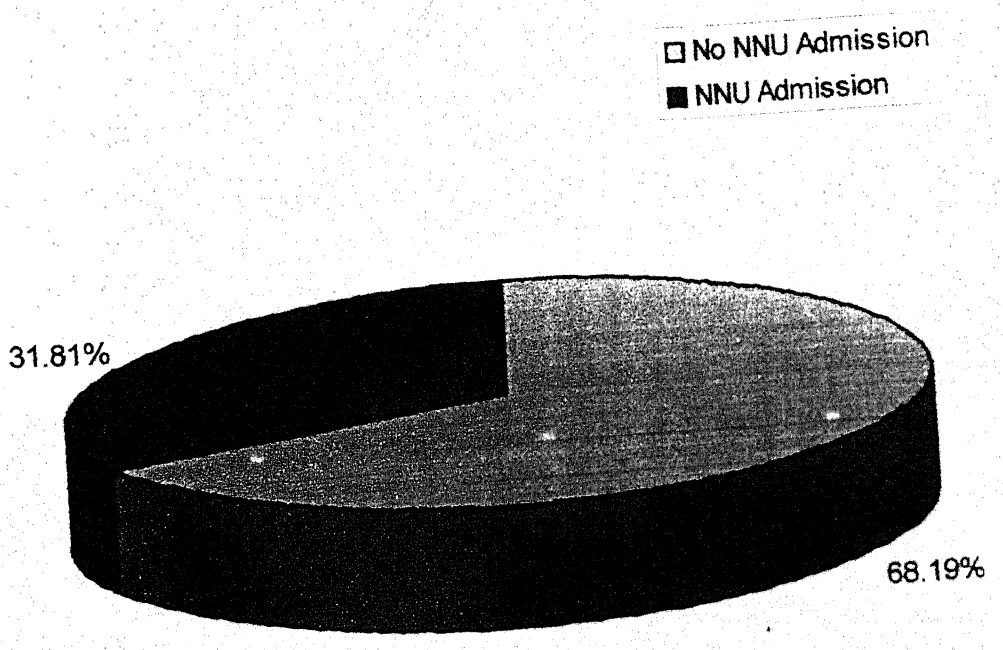
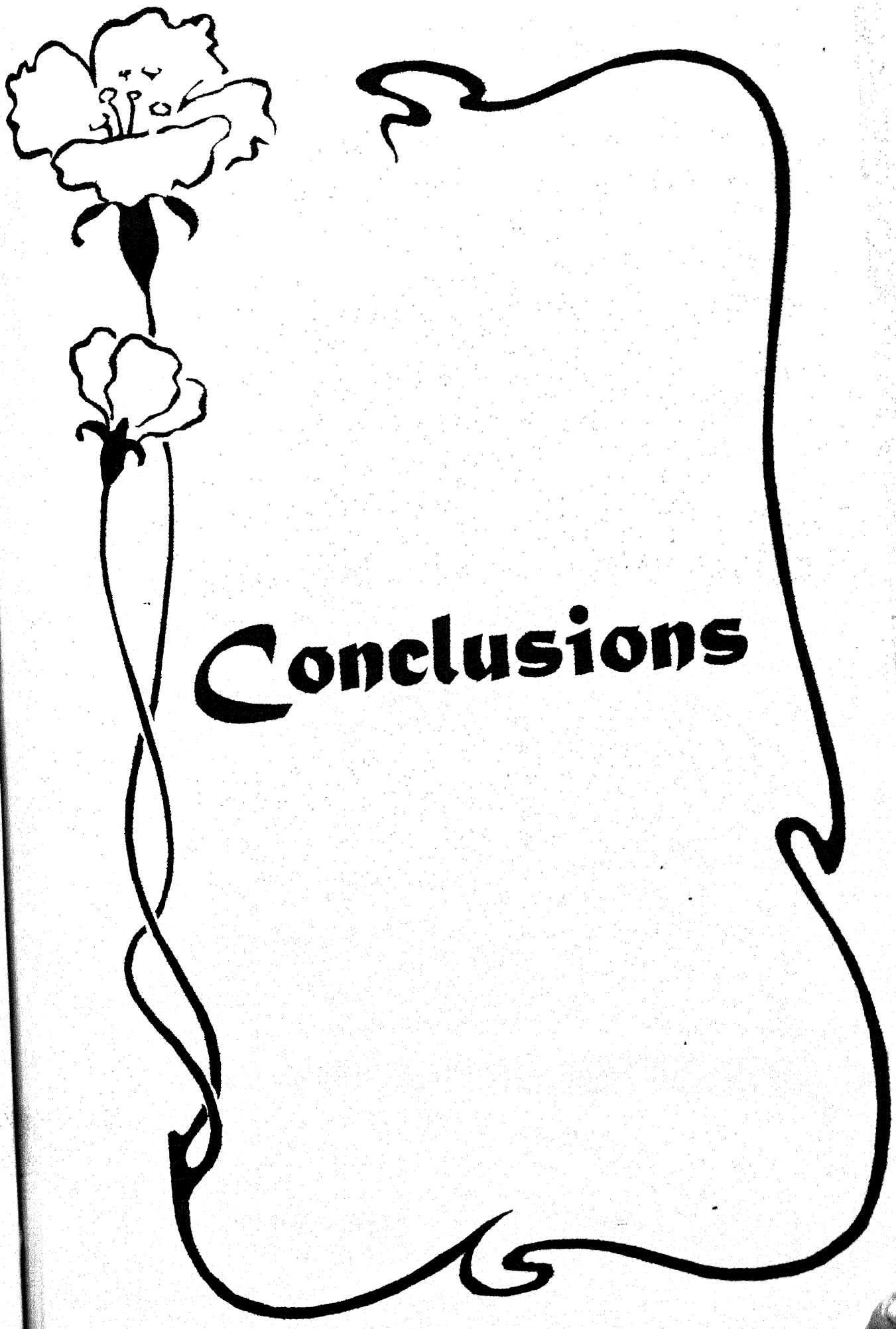


Fig. 10b : NNU ADMISSION IN ABNORMAL NST GROUP (n=44)



Conclusions

Conclusions

The-study ***"Role of Admission test as a Screening procedure for perinatal Outcome"*** was carried out as a prospective blind study in a group of 150 patients in both "high risk" and "low risk pregnancies". These patients were followed in labour room routinely and important points during labour and outcome of pregnancies were compared subsequently and following conclusions were drawn:

1. There were 106 patients (70.67%) showing reactive NST, 28 patients (18.67%) showing equivocal results of NST and 16 (10.67%) patients showing non-reactive non-stress test.

2. When the results of admission test were evaluated age and parity wise, no definite relation would be drawn.

3. Baseline heart rate when seen in study group showed most of the cases (80%) falling in the range *i.e.* 110-150 beats/minute described by F1GO guidelines, 1987.

4. Distribution of result of admission test among patients of different gestational age groups showed slightly more chances of test being abnormal in postdated patients >40 weeks when compared with other patients.

5. There were 99 patients with one or more risk factors. There was more risk of test being abnormal in high risk pregnancies but it was not significant when individual risk factors were compared same conclusion was drawn.

6. Amniotic fluid index was calculated in all patients by ultrasound using four quadrant method. It was statistically established that in patients with AFI <10 cm there were more cases with abnormal admission test, when compared with cases with AFI >10 cm ($p<0.001$).

7. There were greater number of cases developing fetal distress later in labour in group of patients with abnormal admission test ($p<0.01$, specificity 78% NPV 73.5%. Although it appeared during the study that patients with abnormal NST developed fetal distress earlier but it was not statistically significant.

8. Association of appearance of meconium in patients with abnormal admission test was found to be highly significant ($p<.0001$ specificity 78.4% NPV 92.45%, Sensitivity – 68%)

9. It was seen that in patients of abnormal NST there were greater cesarean sections done for fetal distress as compared with patients with reactive admission test but on statistical evaluation it was found to be insignificant.

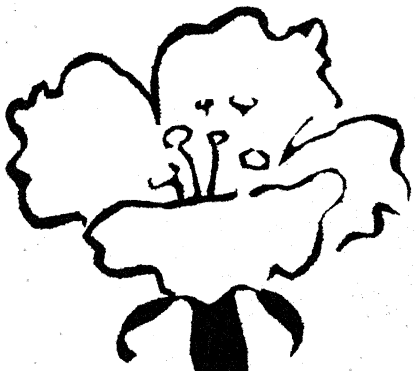
10. When Apgar score of babies at 5 minutes were evaluated it was established that in patients with abnormal admission test greater percentage of babies showed lower apgar scores (sensitivity 66.67% specificity 73.91%, NPV 96.22%,) and more admissions in neonatalogy unit (specificity 76.74%, NPV 93.39% sensitivity 66.67%) ($p<.001$).

12. Perinatal mortality was seen more in cases showing abnormal admission test. (R=5.05).

To conclude we can say that admission test can be used as an important non-invasive method to diagnose the fetal compromise

present at the time of admission in high risk as well as low risk patients in early labour.

It can also categorise patients who would require more strict and vigilant monitoring in the form of continuous electronic fetal monitoring and the ones who can be managed by intermittent auscultation or NST during labour. This will decrease the load of continuous monitoring in all high risk patients and make the obstetrician more careful even in patients with low risk but with abnormal admission test. It can also help them decide when and what timely intervention is to be taken to prevent fetal demise.



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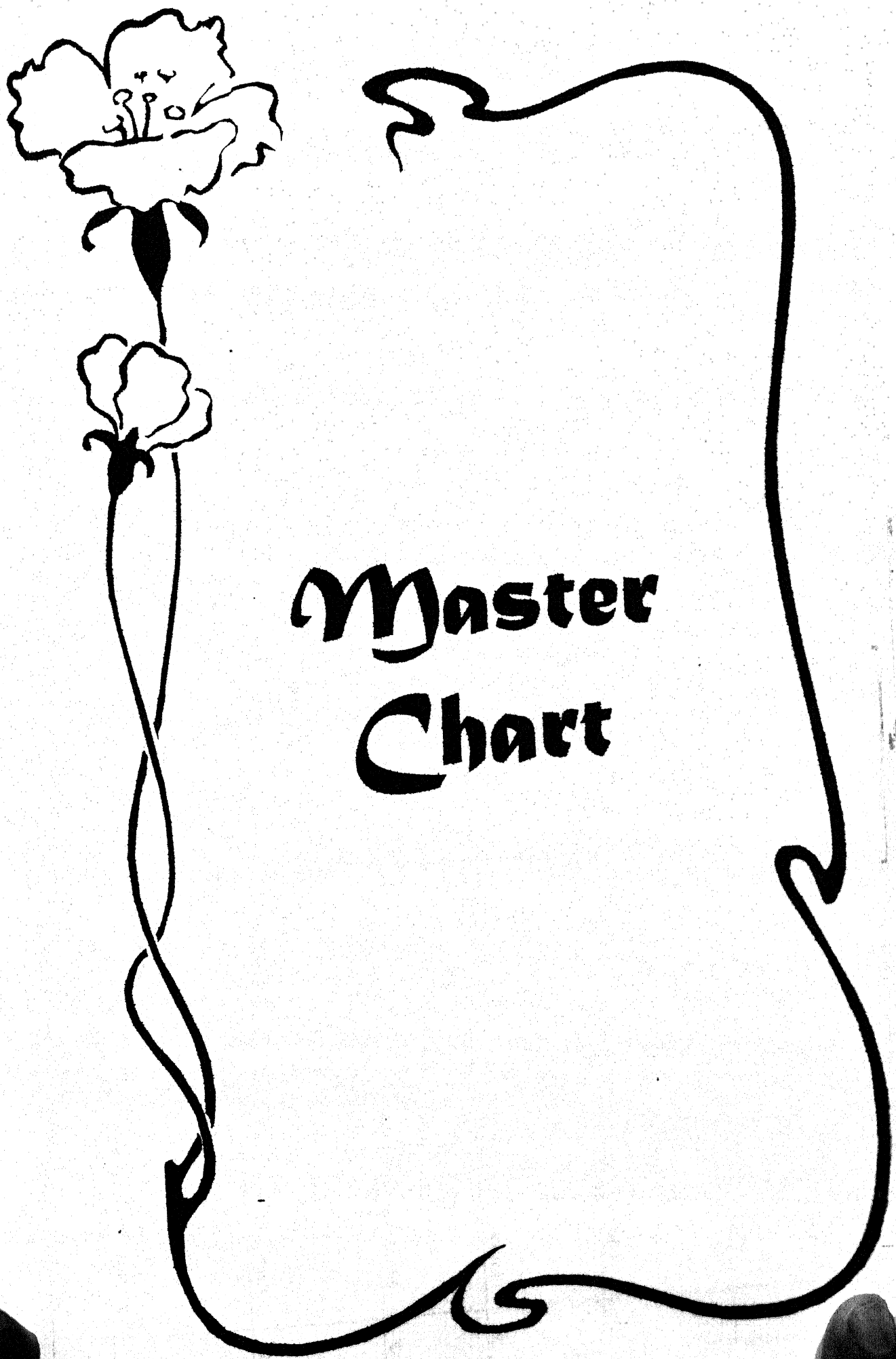

Master Chart

S. No	Name	Age in (yrs)	Parity	No. of live issues	High risk factor	Gestati-onal age	Finding of admission test	Amniotic fluid index	Interval between NST & delivery	Fetal outcome
1.	Poonam	20	P_2+_0	2		36 weeks 3 days	Baseline -144/min Beat to beat variability - 8 Acc – 4 Dec – None Movement –3 Result -Reactive	9.9	12 hrs 10 min	FTND Female 2.5 Kg APGAR (10) at 5 min No mec No cord round neck No NNU adm
2.	Usha	25	P_3+_0	2	Previous LSCS	37 weeks 2 days	Baseline -180/min Beat to beat variability - 30 Acc – None Dec – tnt Movement – 1 **Result – Non reactive**	6	6 hrs	LSCS done for fetal distress Male 3.2 Kg APGAR (10) at 5 min No cord round neck No NNU adm
3.	Sheela	25	P_3+_2	None	Bad obstetric history (Prev. abortion)	38 weeks	Baseline -120/min Beat to beat variability - 20 Acc – None Dec – none Movement – 4 Result – Reactive	12	12 hrs	• LSCS done for fetal distress Male 3.2 Kg APGAR (10) at 5 min No cord round neck No NNU adm
4.	Sarika	27	P_2+_0	2	Previous 2 ISCS Anaemia	36 weeks	Baseline -142/min Beat to beat variability - 20 Acc – 4 Dec – None Movement – 2 Result – Reactive	10	48 hrs	FTND Male 2.75 Kg No mec No cord round neck No NNU adm
5.	Neelam	20	P_0+_2	-	Over due 5 days Prev. abortion	40 weeks 5 days	Baseline -142/min Beat to beat variability - 20 Acc – 3 Dec – None Movement – 3 Result – Reactive	11	21 hrs	FTND Female 3 Kg APGAR (10) at 5 min No mec No cord round neck No NNU adm
6.	Anita	28	P_0+_0	-	-	39 weeks	Baseline -148/min Beat to beat variability - 5 Acc - 2 Dec – None	8	10 hrs	FTND Male 2.8 Kg APGAR (10) at 5 min No mec

							Movement – 1 Result – Reactive			No cord round neck No NNU adm
7.	Sanju	23	P_0+_0	-	-	39 weeks	Baseline -152/min Beat to beat variability - 25 Acc – None Dec – None Movement – 1 **Result -Suspicious**	7	15 hrs	FTND Male 3 Kg APGAR (10) at 5 min No mec No cord round neck No NNU adm
8.	Rekha	18	P_0+_0	-	-	37 weeks	Baseline -140/min Beat to beat variability - 20 Acc – Present Dec – Present (Variable) Movement – None **Result – Suspicious**	10	10 hrs	FTND Female 2.5 Kg APGAR (10) at 5 min No mec **One loop of cord arround neck** No NNU adm
9.	Renu	26	P_0+_0	-	Decreased	39 weeks	Baseline -140/min Beat to beat variability - 15 Acc – 2 Dec – None Movement – 3 Result – Reactive.	12	12 hrs	FTND Female 2.75 Kg APGAR (10) at 5 min No mec No cord round neck No NNU adm
10.	Reena	20	P_0+_0	-	-	38 weeks	Baseline -125/min Beat to beat variability - 10 Acc – Present Dec – Absent Movement – 2 Result – Reactive	11	15 hrs	FTND Female 2.75 Kg APGAR (10) at 5 min No mec One loop of cord arround neck No NNU adm
11.	Seema	19	P_0+_0	-	PET	40 weeks 40 days	Baseline -146/min Beat to beat variability - 25 Acc – 4 Dec – 1 Movement – 4 Result – Reactive	10	10 hrs	LSCS done for fetal distress Female 3.15 Kg APGAR (10) at 5 min No mec No cord round neck No NNU adm

12.	Alka	22	P_0+_0	-	Overdue 2 day decreased fetal movement	40 weeks 2 days	Baseline -126/min Beat to beat variability - 15 Acc – + nt Dec – nt Movement – 3 Result – Reactive	9	24 hrs	LSCS done for fetal distress Female 3 Kg APGAR (8) at 5 min No mec No cord round neck No NNU adm
13.	Kusum	20	P_1+_0	1	-	38 weeks	Baseline -128/min Beat to beat variability - 20 Acc – 1 Dec – None Movement – 4 Result – Reactive	14	20 hrs	FTND Male 3 Kg APGAR (10) at 5 min No mec No cord round neck No NNU adm
14.	Sajjo	25	P_0+_0	-	PET IUGR	39 weeks	Baseline -130/min Beat to beat variability - 15 Acc – 2 Dec – None Movement – 5 Result – Reactive,	16	24 hrs	LSCS done for IUGR with PET Male 2.3 Kg Apgar (10) at 5 min No mec No cord round neck No NNU adm
15.	Rani	20	P_1+_0	1	IUGR	35 weeks	Baseline -146/min Beat to beat variability - 10 Acc – + nt Dec – - nt Movement – 2 Result – Reactive	13	26 hrs	FTND Female – 2.6 Kg APGAR (10) at 5 min No mec No cord round neck No NNU adm
16.	Malti	35	P_1+_0	1	Heart disease (RHD with MS) IUGR	34 weeks	Baseline -142/min Beat to beat variability - 20 Acc – + nt Dec – - nt Movement – 2 Result – Reactive	11	26 hrs	FTND Female – 2.0 Kg APGAR (10) at 5 min No mec No cord round neck No NNU adm
17	Pratiksha	28	P_{0+0}	-	Loss of fetal movements	37 weeks + 2 days	Baseline -140/min Beat to beat variability - 10 Acc – none Dec – none	13	4 hrs	LSCS for loss of fetal movements with fetal distress Male – 2.5 Kg Apgar (8) at 5 min

							Movement – none **Result – suspicious**			**Meconium stained liquor** Once arround NNU adm X 5 days
18	Pratibha	25	P_{0+0}	-	IUGR	38 weeks + 3 days	Baseline -140/min Beat to beat variability - 15 Acc – 3 Dec – none Movement – 3 Result – reactive	9	6 hrs	LSCS for fetal distress Female 3 Kg APGAR (10) at 5 min No mec No cord round neck No NICU adm
19	Sharda	28	P_{1+0}	-	Previous LSCS overdue 7 d.	41 weeks	Baseline -110/min Beat to beat variability - 3 Acc – none Dec –1 Movement – 4 **Result – Non- reactive**	8	3 hrs	LSCS for prev. LSCS with fetal distress Female 3.4 Kg APGAR (5) at 1min (7) at 5 min No mec No cord round neck **NICU adm X 7 days**
20	Chandana	29	P_{0+1}	-	PET IUGR (prev. abortion)	37 weeks	Baseline –130/min Beat to beat variability - 12 Acc – 3 Dec – none Movement – 3 Result – reactive	10	26 hrs	LSCS for PET & fetal distress Male 2.5 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
21	Fameeda	30	P_{1+0}	1	-	38 weeks	Baseline -150/min Beat to beat variability - 12 Acc – 2 Dec – none Movement – 2 Result – reactive	13	6 hrs	FTND Male – 3 Kg APGAR (10) at 5 min No mec No cord No NICU adm
22	Meena	27	P_{1+0}	1	PET overdue 12 days	41 weeks + 5 days	Baseline -150/min Beat to beat variability - 15 Acc – 3 Dec – none Movement – 3 Result – reactive	16	8 hrs	FTND Male – 4Kg APGAR (10) at 5 min No mec No cord No NICU adm

23	Jagjeet Kaur	30	P_{0+0}	-	Overdue 8 days	41 weeks 1 day	Baseline -150/min Beat to beat variability - 12 Acc – 2 Dec – none Movement – 2 Result – reactive	10	14 hrs	LSCS for NPOL with fetal distress Female 4 Kg APGAR (10) at 5 min No mec No cord No NICU adm
24	Kalpana Soni	25	P_{0+0}	-	PET IUGR Oligohydroa mnios overdue 2 day	40 weeks + 2 day	Baseline -140/min Beat to beat variability - 5 Acc – none Dec – none Movement – 2 **Result –Non- reactive**	4	6 hrs	LSCS IUGR with fetal distress Female 1.7 Kg A/S (4)at 1min (6) at 5 min No meconium No cord **Admitted – Expired after 3 days**
25	Anju	25	P_{0+0}	-	-	38 weeks	Baseline -160/min Beat to beat variability - 10 Acc – 1 Dec –2 Movement – 4 **Result –suspicious**	8	10 hrs	FTND Male –2.5 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
26	Jyoti	27	P_{1+0}	1	-	38 weeks + 6 days	Baseline -140/min Beat to beat variability - 12 Acc – 2 Dec – none Movement – 2 Result – reactive	12	24 hrs	FTND Male – 3 Kg APGAR (8) at 5 min **Thin meconium** No cord No NICU adm
27	Jaidevi	29	P_{3+0}	2	IUGR Ologohy=dro amnos overdue 5 days	40 weeks + 5 days	Baseline -146/min Beat to beat variability - 10 Acc – none Dec –2 Movement – 4 **Result –suspicious**	7	2 hours	FTND Female – 2.7 Kg APGAR (10) at 5 min No mec No cord No NICU adm
28	Stuti	21	P_{0+0}	-	Oligohyd	37 weeks	Baseline -152/min Beat to beat variability - 3 Acc – none Dec – none	6	6 hrs	LSCS for NPOL with fetal distress Female 2.3 Kg APGAR (8) at 5 min

							Movement – 4 **Result – Non- reactive**			Thin meconium No cord No NICU adm
29	Sapna	28	P_{0+2}	-	Overdue 14 day 2 prev. abortion	42 weeks	Baseline -160/min Beat to beat variability - 8 Acc – 2 Dec – none Movement – 1 **Result –suspicious**	12	10 hrs	FTND Male – 2.5 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
30	Mamta	25	P_{2+0}	2	Previous 2 LSCS	37 weeks + 4 day	Baseline -120/min Beat to beat variability - 3 Acc – none Dec – none Movement – none **Result –Non- reactive**	9	2 hrs	LSCS for prev 2 LSCS Male 2.3 Kg APGAR (6) at 5 min **Thick meconium stained** No cord **NICU adm X 3 days**
31	Vandana	24	P_{1+0}	1	Previous LSCS Rh neg preg	40 weeks	Baseline -130/min Beat to beat variability - 12 Acc – 3 Dec – none Movement – 3 Result –reactive	14	16 hrs	LSCS for prev LSCS with fetal distress Male 3 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
32	Jaikunear	22	P_{1+0}	1	Previous LSCS overdue 9 days decreased fetal movements	42 weeks + 2 day	Baseline -150/min Beat to beat variability - 15 Acc – 5 Dec – none Movement – 3 Result –reactive	11	5 hrs	LSCS for prev LSCS with overdue 9 days Female 3 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
33	Rehana	22	P_{1+1}	1	Previous LSCS previous abortion	40 weeks + 4 day	Baseline -138/min Beat to beat variability -10 Acc – 6 Dec –1 Movement – 2 Result reactive	16	28 hrs	FTND Male – 2.75 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm

34	Rekha	26	P_{0+0}	-	-	38 weeks	Baseline -140/min Beat to beat variability -25 Acc – none Dec –none Movement – 1 **Result –suspicious**	11	3.5 hrs	FTND Feale – 2.25 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
35	Manku	20	P_{0+0}	-	Diminished fetal movements	38 weeks	Baseline -150/min Beat to beat variability -18 Acc – 7 Dec –none Movement – 1 Result –reactive	10	9 hrs	LSCS for loss of fetal movements Female 2.4 Kg APGAR (10) at 5 min No meconium **Cord once around neck** No NICU adm
36	Sheela	26	P_{0+0}	-	-	37 weeks	Baseline -150/min Beat to beat variability -15 Acc – +nt Dec –none Movement – 2 Result –reactive	10	20 hrs	FTND Male – 3.6 Kg APGAR (8) at 5 min No meconium No cord around neck No NICU adm
37	Reena	26	P_{0+0}	-	Decreased fetal movements overdue 6 days	40 weeks + 6 day	Baseline -146/min Beat to beat variability -14 Acc – none Dec –none Movement – none **Result –suspicious**	9	2.5 hrs	LSCS for decreased fetal movements Female 2.6 Kg APGAR (10) at 5 min **Mec - +nt** No cord round neck No NICU adm
38	Kanti	20	P_{1+0}	None	Previous LSCS none alive	38 weeks + 1 day	Baseline -154/min Beat to beat variability -25 Acc – none Dec –none Movement – 1 **Result –suspicious**	8	5 hrs	FTND Male – 2.8 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
39	Nandi	26	P_{0+0}	-	Overdue 4 days	40 weeks + 4 days	Baseline -140/min Beat to beat variability -10 Acc – none Dec – none	9	12.5 hrs	FTND Female – 2.5 Kg APGAR (10) at 5 min No meconium

							Movement – none **Result –suspicious**			No cord round neck No NICU adm
40	Shashi	25	P_{1+2}	1	Previous 2 abortions	38 weeks	Baseline -146/min Beat to beat variability -15 Acc – + Dec – Movement – 2 Result –reactive	12	2 hrs	FTND Male – 3 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
41	Sushama	22	P_{0+3}	-	3 abortion	36 weeks 4 days	Baseline -140/min Beat to beat variability -8 Acc + Dec – Movement – 4 Result –reactive	16	10 hrs	LSCS done for BOH with high floating head Male 2.8 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
42	Kusma	25	P_{6+0}	4	-	37 weeks	Baseline -148/min Beat to beat variability -10 Acc + Dec – Movement – 4 Result –reactive	11	5.5 hrs	FTND Female – 3.6 Kg APGAR (8) at 5 min No meconium No cord round neck No NICU adm
43	Krishna	23	P_{0+0}	-	IUGR Oligohyd-romnios	38 weeks 2 days	Baseline -150/min Beat to beat variability -4 Acc – none Dec –none Movement – 2 **Result –suspicious**	4	1.5 hrs	LSCS done for fetal distress Male 1.5Kg APGAR (6) at 5 min **Mec (+)** **Baty admitted in NICU** **Expired after 24 hours because of RDS**
44	Anu	26	P_{1+0}	1	Previous LSCS overdue 13 days decreased fetal movements	41 weeks 6 days	Baseline -136/min Beat to beat variability -25 Acc – 4 Dec –none Movement – 2 Result –reactive	10	23 hrs	LSCS done for prev LSCS Male 2.8 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
45	Rukmini	40	P_{2+1}	2	Previous abortion	40 weeks + 5 days	Baseline -140/min Beat to beat variability -10	9	8 hrs	FTND Female 3 Kg

					elderly		Acc – 3 Variable dec –3 Movement – 2 **Result –suspicious**			APGAR (10) at 5 min No meconium No cord round neck No NICU adm
46	Sarita	26	P_{1+0}		Previous LSCS none alive	37 weeks	Baseline –142/min Beat to beat variability -5 Acc – none Dec –2 Movement – none **Result –Non-reactive**	9	26 hrs	LSCS done for inadequate pelvis with fetal distress Female 2.15 Kg APGAR (10) at 5 min No cord round neck **Mecon (+)** No NICU adm
47	Sabeena	24	P_{1+0}	None	Previous still birth Eclamypsia in previous pregnancy	39 weeks	Baseline -150/min Beat to beat variability -10 Acc – 2 Dec –none Movement – 2 Result –reactive	12	20 hrs	LSCS done for BOH with acute fetal distress Female 2.5Kg APGAR (10) at 5 min No Mec No cord round neck No NICU adm
48	Reeta	20	P_{1+0}	1	Prev. LSCS	40 weeks + 5 days	Baseline -136/min Beat to beat variability -10 Acc – 4 Dec –none Movement – 2 Result –reactive	12	14 hrs	FTND Male – 3.2 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
49	Sangeeta	30	P_{2+0}	2	Bronchial asthma decreased fetal movements	36 weeks	Baseline -136/min Beat to beat variability -10 Acc – 2 Dec –none Movement – none Result –reactive	12	6 hrs	FTND Male – 2.25 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
50	Vidya	35	P_{0+0}	-	PET Brech elderly primi	38 weeks	Baseline -138/min Beat to beat variability -5 Acc – 7 Dec –none Movement – 2 Result –reactive	10	48 hrs	LSCS done for primi breech Female 2.9 Kg APGAR (10) at 5 min **Mec (+)** No cord round neck No NICU adm

51	Rama	20	P_{1+0}	-	Previous still birth	39 weeks + 3 days	Baseline -140/min Beat to beat variability -15 Acc (+) Dec (-) Movement – 2 Result –reactive	11	20 hrs	FTND Female – 2.75 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
52	Nagina	28	P_{2+1}	2	16 days overdue prev. 2 abortion	42 weeks +2 days	Baseline -138/min Beat to beat variability -10 Acc – 2 Dec –none Movement – 1 Result –reactive	7	24 hrs	LSCS for overdue Male 3.5Kg APGAR (10) at 5 min No mec Cord round neck once No NICU adm
53	Seema	27	P_{3+0}	1	Previous 2 still births	36 weeks +4 days	Baseline -136/min Beat to beat variability -15 Acc –none Dec –none Movement – none **Result –suspicious**	12	**6 hrs**	LSCS for BOH Male 2.9 Kg APGAR (10) at 5 min **Meconium (+)** No cord round neck No NICU adm
54	Savitri	29	P_{0+0}	-	Infertility treated	39 weeks +6 days	Baseline -150/min Beat to beat variability -5 Acc – 3 Dec –none Movement – 2 Result –reactive	11	4.5hrs	LSCS done for acute fetal distress Male 2.75 Kg APGAR (10) at 5 min No mec **Cord (+) once** No NICU adm
55	Meera	32	P_{0+2}	-	2 abortion overdue 3 days	40 weeks + 3 days	Baseline -140/min Beat to beat variability -5 Acc – 2 Dec –1 Movement – 3 **Result –suspicious**	6	2 hrs	LSCS for fetal distress Female 2.9 Kg APGAR (10) at 5 min **Mec (+)** No cord round neck No NICU adm
56	Karma	27	P_{0+0}	-	PET	37 weeks +	Baseline -142/min	8	16 hrs	FTND

						6 days	Beat to beat variability -15 Acc – none Dec –none Movement – 2 Result –reactive			Male – 2. 5 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
57	Sheela	25	P_{0+0}	-	-	38 weeks	Baseline -134/min Beat to beat variability -10 Acc – 2 Dec –none Movement – 3 Result –reactive	12	15 hrs	FTND Female – 2.75 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
58	Snehlata	25	P_{0+0}	-	PET	37 weeks + 2 days	Baseline -162/min Beat to beat variability -5 Acc – none Dec –5 Movement – 1 **Result –Non-reactive**	12	6 hrs	FTND Male – 2. 5 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
59	Geeta	28	P_{1+0}	1	Overdue 7 days anaemia	41 weeks	Baseline -140/min Beat to beat variability -10 Acc – 3 Dec –none Movement – 3 Result –reactive	10.5	10 hrs	FTND Male – 2. 5 Kg APGAR score 10 No meconium No cord No NICU adm
60	Suman	24	P_{0+0}	-	-	37 weeks	Baseline -138/min Beat to beat variability -5 Acc – 2 Dec –none Movement – 2 Result –reactive	10	5 hrs	FTND Female – 2.75 Kg APGAR (10) No meconium **Cord (+) once** No NICU adm
61	Leela	28	P_{0+0}	-	IUGR	39 weeks	Baseline -142/min Beat to beat variability -15 Acc – 4 Dec –1 Movement – 1 Result –reactive	6	26 hrs	FTND Female – 2.2 Kg APGAR (6) at 5 min **Thickmeconium** **Sec. apnea – admitted in NICU** **I/V, O2, Abs** **Multiple anomalies**

										expired after 24 hour
62	Vineeta	20	P_{0+0}	-	-	38 weeks + 4 days	Baseline -160/min Beat to beat variability -10 Acc + Dec (+) Movement –none **Result –suspicious**	10	20 hrs	FTND Male – 3 Kg APGAR (10) at 3min No mec (-) No cord round neck No NICU adm
63	Anita	29	P_{1+0}	-	Prev. LSCS Anaemia Prev. still birth	38 weeks + 6 days	Baseline -135/min Beat to beat variability -5 Acc (+) Dec absent Movement – 3 Result –reactive	12	17 hrs	LSCS for fetal distress Male 3.2 Kg APGAR (10) at 5 min **Thick meconium present** **No cord round neck** **NICU adm X 3 days**
64	Reshma	24	P_{0+0}	-	Overdue 7 days	41 weeks	Baseline -144/min Beat to beat variability -18 Acc –(+) Dec (+) variable Movement – 4 Result –reactive	8	2 hrs	LSCS done for fetal distress Male 2. 5 Kg APGAR (10) at 5 min No mec Cord round neck present No NICU adm
65	Neelu	26	P_{0+0}	-	-	37 weeks + 4 days	Baseline -140/min Beat to beat variability -12 Acc – 3 Dec –3 Movement – none Result –reactive	9	16 hrs	FTND Male – 3.5 Kg APGAR (10) at 5 min No mec No cord round neck No NICU adm
66	Roshni	22	P_{0+0}	-	PET	39 weeks	Baseline -130/min Beat to beat variability -10 Acc 2 Dec –none Movement – 3 Result –reactive	10	20 hrs	LSCS for NPOL with fetal distress Female 3. 5 Kg APGAR (10) at 5 min No mec No cord No NICU adm
67	Munni Devi	30	P_{3+0}	3	-	38 weeks	Baseline -146/min Beat to beat variability -12 Acc 2 Dec none	9	4 hrs	FTND Male 2.5 Kg APGAR (10) at 5 min No mec

							Movement – 3 Result –reactive			No cord No NICU adm
68	Sunita	25	P_{1+0}	1	-	38 weeks 3 days	Baseline -150/min Beat to beat variability -10 Acc – 4 Dec –none Movement – 4 Result –reactive	12	10 hrs	FTND Female – 2.25 Kg APGAR (6) at 5 min **Meconium present** No cord **NICU admission X 5 days**
69	Malti	27	P_{1+1}	1	PET Prev. LSCS prev. abortion	38 weeks + 5 days	Baseline -146/min Beat to beat variability -12 Acc – 2 Dec –none Movement – 2 Result –reactive	12	20 hrs	LSCS for prev LSCS with fetal distress Female 3.2 Kg APGAR (10) at 5 min No mec No cord No NICU adm
70	Shobha	20	P_{0+0}	-	-	38 weeks	Baseline -120/min Beat to beat variability -3 Acc – none Dec –none Movement – 2 **Result – Non-reactive**	8	6 hrs	LSCS for fetal distress Male 2. 6 Kg APGAR (6) at 5 min **Thick meconium** No cord **NICU adm X 7 days**
71	Kiran	25	P_{1+0}	1	-	37 weeks + 4 days	Baseline -150/min Beat to beat variability -10 Acc – 4 Dec –none Movement – 4 Result –reactive	11	12 hrs	LSCS for fetal distress Male 2. 7Kg APGAR (9) at 5 min No meconium **Cord once round neck** No NICU adm
72	Rani	21	P_{0+0}	-	Rh neg	39 weeks + 2 days	Baseline -120/min Beat to beat variability -12 Acc – 4 Dec –none Movement – 5 Result –reactive	10	16 hrs	FTND Female – 3 Kg APGAR (10) at 6 min No meconium No cord No NICU adm
73	Sangeeta	28	P_{1+1}	1	Prev. LSCS	38 weeks +	Baseline -150/min	10	14 hrs	LSCS done for prev LSCS

					diminished fetal movements prev. abortion	1 day	Beat to beat variability -12 Acc – 5 Dec –none Movement – 5 Result –reactive			with fetal distress Female 2.3 Kg APGAR (10) at 5 min No mec **Cord once around neck** No NICU adm
74	Sahana	22	P_{0+0}	-	Overdue 3 days	40 weeks + 3 days	Baseline -140/min Beat to beat variability -12 Acc – 3 Dec –1 Movement – 3 Result –reactive	12	10 hrs	FTND Male – 2.5 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
75	Nazneen	25	P_{0+0}	-	Overdue 8 days oligohydramnios	41 weeks + 1 day	Baseline -140/min Beat to beat variability -15 Acc – 2 Dec –2 variable Movement – 2 **Result –suspicious**	6	22 hrs	LSCS for NPOL Female 3.2Kg APGAR (10) at 5 min **Thick meconium** **Cord once round neck** **NICU adm X 5 days**
76	Rani	26	P_{0+0}	-	-	38 weeks + 2 days	Baseline -146/min Beat to beat variability -12 Acc – 3 Dec –none Movement – 3 Result –reactive	10	16 hrs	FTND Male – 3.o Kg APGAR (10) at 5 min No meconium No cord No NICU adm
77	Pratibha	26	P_{0+0}	-	-	37 weeks	Baseline -136/min Beat to beat variability -15 Acc – 3 Dec –none Movement – 3 Result –reactive	12	28 hrs	FTND Female – 3Kg APGAR (5) at 5 min No thick meconium No cord No NICU adm
78	Moola bai	26	P_{0+0}	-	-	39 weeks	Baseline –150/min Beat to beat variability -10 Acc – none Dec –none Movement – 5 Result reactive	7.5	6 hrs	LSCS for contracted pelvis & fetal distress Female 3.0Kg APGAR (5) at 5 min **Thick meconium** No cord **NICU adm X 5 days**
79	Seema	28	P_{1+1}	1	-	38 weeks +	Baseline -140/min	10	8 hrs	FTND

						2 days	Beat to beat variability -12 Acc – 3 Dec –none Movement – 3 Result –reactive			Male – 3.5 Kg APGAR (10) at 5 min No mec No cord No NICU adm
80	Sushmita	25	P_{1+0}	1	PET	38 weeks + 5 days	Baseline -150/min Beat to beat variability -10 Acc – 3 Dec –none Movement – 3 Result –reactive	12	13 hrs	LSCS for fetal distress Male 2.5Kg APGAR (10) at 5 min No meconium No cord No NICU adm
81	Pushpa	24	P_{1+0}	1	Overdue 7 days	41 weeks	Baseline -160min Beat to beat variability -20 Acc – none Dec –present Movement – 4 Result –reactive	8	21 hrs	LSCS done for overdue with free floating head Male 2.6 Kg APGAR (8) at 5 min No meconium No cord No NICU adm
82	Shobha	26	P_{1+0}	None	Prev. still birth	40 weeks	Baseline –142/min Beat to beat variability -10 Acc – +nt Dec – - nt Movement – 6 Result –reactive	9	17 hrs	FTND Male – 2.6 Kg APGAR (10) at 5 min No mec No cord No NICU adm
83	Alpana	23	P_{0+0}	-	IUGR	36 weeks + 6 days	Baseline -142/min Beat to beat variability -10 Acc – +nt Dec – -nt Movement – 2 Result –reactive	8	14 hrs	LSCS done for fetal distress Male 2.8 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
84	Laxmi	22	P_{0+0}	-	IUGR Anaemia	38 weeks	Baseline -162/min Beat to beat variability -8 Acc – none Dec –+nt Movement – none **Result – Non-reactive**	12	1.5 hrs	FTND Male – 2.25 Kg APGAR (10) at 5 min **Thick mec (+)** No cord round nec **NICU adm X 7 days**
85	Mrinal	20	P_{1+1}	1	Polyhydramnios prev.	37 weeks	Baseline -142/min Beat to beat variability -15	22	16 hrs	LSCS for contracted pelvis Male 3.5Kg

					abortion		Acc – +nt Dec – -nt Movement – 8 Result –reactive			APGAR (10) at 5 min No meconium No cord No NICU adm
86	Mani	25	P_{0+0}	-	Overdue 8 days	41 weeks + 1 day	Baseline -150/min Beat to beat variability -15 Acc – 3 Dec –none Movement – 5 Result –reactive	11	12 hrs	FTND Female – 2.75 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
87	Pana	28	P_{1+1}	-	Previous anencephaly still birth	37 weeks + 2 day	Baseline -142/min Beat to beat variability -5 Acc – none Dec – +nt Movement – 1 **Result – Non-reactive**	10	4 hrs	LSCS done for acute fetal distress **Male 2.8 Kg** APGAR (10) at 5 min No meconium **Cord twice round neck** No NICU adm
88	Sonam	23	P_{0+0}	-	-	38 weeks + 1 day	Baseline -148/min Beat to beat variability -10 Acc – (+) Dec – (+) Movement – 3 Result –reactive	11	12 hrs	FTND Female – 2. 5 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
89	Meera	24	P_{0+0}	-	Overdue 3 days	40 weeks + 3 days	Baseline -150/min Beat to beat variability -20 Acc – (+) Dec –none Movement – 6 Result –reactive	10	5 hrs	FTND Female – 2.75 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
90	Sanno	22	P_{0+0}	-	Infertility lead IUGR severe oligohydromnios	37 weeks +2 day	Baseline -150/min Beat to beat variability -25 Acc – (+) Dec – (+) Movement – 1 **Result –suspicious**	2	13 hrs	LSCS done for acute fetal distress Male 2.5 Kg APGAR (8) at 5 min **Mec (+)** **NICU admission** **Baby expired 3rd day**
91	Neelu	28	P_{3+0}	3	Rh –ve IUGR	40 weeks	Baseline -130/min Beat to beat variability -20 Acc – none	12	10 hrs	LSCS for fetal distress Female2.8 Kg APGAR (8) at 5 min

							Dec –none Movement – none **Result –suspicious**			**Meconium (+)** No Cord No NICU adm
92	Anju	27	P_{1+0}	1	-	37 weeks + 5 days	Baseline -140/min Beat to beat variability -5 Acc – 4 Dec –none Movement – 5 Result –reactive	10	2 hrs	LSCS done for fetal distress Female 3.2 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
93	Roshan	28	P_{2+0}	2	Prev. LSCS PET Tranverse Lie	37 weeks	Baseline -160/min Beat to beat variability -15 Acc – (+) Dec – Movement – 6 Result –reactive	10	26 hrs	LSCS done for PET & transverse lie Male 2.8 Kg APGAR (10) at 5 min No meconium No Cord round neck No NICU adm
94	Heera	26	P_{0+0}	-	-	38 weeks	Baseline -152/min Beat to beat variability -20 Acc + Dec (variable) (+) Movement – 1 **Result –suspicious**	11	72 hrs	FTND Female – 2.8Kg APGAR (10) at 5 min No meconium No cord No NICU adm
95	Anjali	22	P_{0+0}	-	-	37 weeks + 2 days	Baseline -130/min Beat to beat variability -10 Acc – 2 Dec – -nt Movement – 4 Result –reactive	12	12 hrs	FTND Male – 2.2 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
96	Indira	23	P_{1+0}	1	Overdue 8 days	41 weeks + 1 day	Baseline -142/min Beat to beat variability -18 Acc – (+) Dec – (-) Movement – 6 Result –reactive	10	15 hrs	FTND Male – 2.8 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
97	Vimla	20	P_{0+0}	-	-	37 weeks + 4 days	Baseline -140/min Beat to beat variability -10 Acc – 2	8	26 hrs	LSCS for fetal distress Male 3 Kg APGAR (10) at 5 min

							Dec –none Movement – 3 Result –reactive			No meconium No cord round neck No NICU adm
98	Nafisa	26	P_{0+2}	-	Previous 2 abortions PET	40 weeks	Baseline -142/min Beat to beat variability -10 Acc – +nt Dec –none Movement – 6 Result –reactive	14	20 hrs	LSCS for NPOL with fetal distress Female 2.6 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
99	Sita	24	P_{1+0}	-	Overdue 6 days	40 weeks + 6 days	Baseline -158/min Beat to beat variability -10 Acc – none Dec –none Movement – none **Result –suspicious**	8	10 hrs	FTND Female – 2.65 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
100	Sundari	29	P_{1+0}	1	-	38 weeks	Baseline -150/min Beat to beat variability -16 Acc – +nt Dec –none Movement – 5 Result –reactive	10	4 hrs	FTND Male – 3.0 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
101	Sushila	26	P_{0+0}	-	-	37 weeks + 6 days	Baseline -140/min Beat to beat variability -10 Acc – +nt Dec – -nt Movement – 6 Result –reactive	10	2 hrs	LSCS done for fetal distress Female 2 Kg APGAR (10) at 5 min **Meconium stained liquor** **Cord around neck** No NICU adm
102	Maggo	28	P_{2+1}	1	Previous still birth	37 weeks + 1 day	Baseline -146/min Beat to beat variability -14 Acc – +nt Dec –nt Movement – 4 Result –reactive	11	45 hrs	FTND Female – 2.75 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
103	Sudha	26	P_{1+0}	1	Overdue 12 days	41 weeks + 5 days	Baseline -138/min Beat to beat variability -10 Acc – +nt	8	24 hrs	FTND Male – 2.6 Kg APGAR (10) at 5 min

							Dec –none Movement – 3 Result –reactive			No meconium No cord round neck No NICU adm
104	Sashi	24	P_{0+0}	-	-	38 weeks	Baseline -140/min Beat to beat variability -15 Acc – +nt Dec –nt Movement – 2 Result –reactive	15	4 hrs	FTND Male – 3.2 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
105	Ramwati	24	P_{0+0}	-	Mild MS type II, placenta praevida (posterior)	36 weeks + 2 days	Baseline -145/min Beat to beat variability -10 Acc – + nt Dec – nt Movement – 4 Result –reactive	10	8 hrs	FTND Female – 2.0 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
106	Aparna	20	P_{0+0}	-	-	38 weeks + 2 days	Baseline -142/min Beat to beat variability -12 Acc – +nt Dec –nt Movement – 2 Result –reactive	11	6 hrs	FTND Female – 2.6 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
107	Gajra	26	P_{2+0}	2	-	39 weeks	Baseline -138/min Beat to beat variability -14 Acc – +nt Dec –none Movement – 3 Result –reactive	12	12 hrs	FTND Female – 2.7Kg APGAR (10) at 5 min No meconium No cord No NICU adm
108	Sampat	22	P_{1+0}	1	Decreased fetal movements RHD with MS withMR (valvotomy done)	36 weeks + 6 days	Baseline -164/min Beat to beat variability -8 Acc – none Dec –3 Movement – none **Result – Non-reactive**	10	1.5 hrs	LSCS done for acute fetal distress Male 2.8Kg APGAR (10) at 5 min **Thick meconium present** No cord No NICU adm
109	Nisha	20	P_{0+0}	-	-	38 weeks	Baseline -138/min Beat to beat variability -15 Acc +nt	12	15 hrs	FTND Female – 2.3 Kg APGAR (10) at 5 min

							Dec –nt Movement – 4 Result – reactive			**Thick meconium present** No cord No NICU adm
110	Fatima	28	P_{1+0}	1	Prev. LSCS overdue 2 days	40 weeks + 2 days	Baseline -148/min Beat to beat variability -8 Acc – 3 Dec –none Movement – 2 Result –reactive	8	24 hrs	LSCS for prev LSCS with fetal distress Female 2.8 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
111	Suman	25	P_{2+0}	2	IUGR	36 weeks	Baseline -142/min Beat to beat variability -8 Acc – 3 Dec –none Movement – 2 Result –reactive	14	16 hrs	FTND Male – 1.5 Kg APGAR (4) at 5 min No mec No cord NICU adm for RDS Expired after 48 hours
112	Radha	20	P_{0+0}	-	-	39 weeks + 4 days	Baseline -140/min Beat to beat variability -10 Acc – +nt Dec –nt Movement – 3 Result –reactive	11	4 hrs	LSCS for contracted pelvis Female 3.0Kg APGAR (10) at 5 min No meconium No cord No NICU adm
113	Alti devi	26	P_{0+1}	-	Jaundice oligohydrmnios prev. abortion	36 weeks	Baseline -120/min Beat to beat variability -3 Acc – none Dec –none Movement – none **Result – Non-reactive**	6	10 hrs	FTND Female – 2.1Kg APGAR (5) at 5 min **Thick meconium present** No cord **NICU adm Expired after 12 hrs**
114	Shrija	25	P_{1+0}	1	PET	37 weeks + 5 days	Baseline -150/min Beat to beat variability -8 Acc – 1 Dec –none Movement – 3 Result –reactive	10	6 hrs	LSCS for PET with fetal distress Male 2.75 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
115	Ragini	32	P_{1+1}	1	Previous	37 weeks +	Baseline -160/min	7	14 hrs	LSCS for fetal distress

					LSCS PET	2 days	Beat to beat variability -25 Acc – 3 Dec –2 Movement – 2 **Result –suspicious**			Male 2.2 Kg APGAR (6) at 5 min **Thick meconium** No cord **NICU adm X 5 days**
116	Shikha	23	P_{0+0}	-	-	39 weeks + 5 days	Baseline -150/min Beat to beat variability -12 Acc – 3 Dec –none Movement – 3 Result –reactive	11	8 hrs	FTND Male – 3.5 Kg APGAR (10) at 5 min No meconium **Cord once round neck** No NICU adm
117	Ahilya	24	P_{0+1}	-	-	37 weeks	Baseline -130/min Beat to beat variability -12 Acc – 2 Dec –none Movement – 2 Result –reactive	11	22 hrs	LSCS for fetal distress Male 2.4 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
118	Lakshmi	26	P_{0+0}	-	Overdue 3 days	40 weeks + 3 days	Baseline -110/min Beat to beat variability -4 Acc – none Dec –none Movement – 2 **Result – Non-reactive**	9	6 hrs	LSCS for NPOL Male 2.5 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
119	Sita devi	26	P_{0+0}	-	Heart disease (MS with MR)	38 weeks + 2 days	Baseline -152/min Beat to beat variability -15 Acc – 4 Dec –none Movement – 4 Result –reactive	10	12 hrs	FTND Female – 2.5 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
120	Kumkum	26	P_{1+0}	1	-	39 weeks	Baseline -152/min Beat to beat variability -10 Acc – 2 Dec –none Movement – 2 Result –reactive	11	8 hrs	FTND Female – 3Kg APGAR (10) at 5 min No meconium **Cord once round neck** No NICU adm
121	Shaheen	23	P_{0+0}	-	PET	37 weeks	Baseline -130/min Beat to beat variability -12 Acc – 2	11	20 hrs	FTND Female – 2.3 Kg APGAR (10) at 5 min

							Dec –none Movement – 2 Result –reactive			No meconium No cord No NICU adm
122	Rajkumari	28	P_{2+0}	2	Prev. LSCS	38 weeks + 4 days	Baseline -130/min Beat to beat variability -8 Acc – 1 Dec –none Movement – 3 Result –reactive	7	11 hrs	LSCS for prev LSCS Female 2.3 Kg APGAR (6) at 1 min (10) at 5 min No meconium **Cord once round neck** **NICU adm X 5 days**
123	Chaaya	22	P_{1+0}	1	-	38 weeks	Baseline -160/min Beat to beat variability -8 Acc – 1 Dec –1 Movement – 3 **Result –suspicious**	9	7 hrs	FTND Female – 2.5 Kg APGAR (9) at 5 min No meconium No cord No NICU adm
124	Mona	22	P_{1+0}	1	-	39 weeks	Baseline -130/min Beat to beat variability -12 Acc – 3 Dec –none Movement – 3 Result –reactive	9.5	22 hrs	LSCS fa N POL with fetal distress Male – 3 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
125	Kamla	27	P_{0+0}	-	-	38 weeks +2 days	Baseline -152/min Beat to beat variability -12 Acc – 2 Dec –none Movement – 2 Result –reactive	10	13 hrs	FTND Male – 2.7 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
126	Bhoomika	26	P_{0+0}	-	Overdue 5 days loss of fetal movements	40 weeks + 5 days	Baseline -150/min Beat to beat variability -15 Acc – 4 Dec –none Movement – 4 Result –reactive	12	4 hrs	LSCS for loss of fetal movements Female 2.6 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
127	Jaidevi	24	P_{1+0}	1	-	37 weeks	Baseline -160/min Beat to beat variability 15	11	16 hrs	LSCS for free floating head Male 3 Kg

							Acc – 3 Dec –none Movement – 3 Result –reactive			APGAR (10) at 5 min No meconium **Cord twice round neck** No NICU adm
128	Sonia	30	P_{1+0}	1	-	37 weeks + 5 days	Baseline -140/min Beat to beat variability -10 Acc – 2 Dec –none Movement – 2 Result –reactive	9	8 hrs	FTND Male – 2.6 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
129	Roma	20	P_{0+0}	-	-	39 weeks +2 days	Baseline -140/min Beat to beat variability -10 Acc – 2 Dec –none Movement – 2 Result –reactive	11	17 hrs	FTND Female – 2.5 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
130	Gayatri	31	P_{1+1}	1	Overdue 11 days oligohydromnios IUGR	41 weeks +4 days	Baseline -150/min Beat to beat variability -15 Acc – 5 Dec –none Movement – 5 Result –reactive	4.5	3 hrs	LSCS for overdue with oligo with fetal distress Female 2 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
131	Zareena	30	P_{0+1}	-	Overdue 10 days PET Prev. abortion	41 weeks +3 days	Baseline -156/min Beat to beat variability -8 Acc – none Dec –none Movement – 2 **Result –suspicious**	13	5 hrs	LSCS for PET & overdue Female 3 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
132	Shabana	26	P_{0+0}	-	-	39 weeks +6days	Baseline -140/min Beat to beat variability -12 Acc – 2 - Dec –1 Movement – 3 Result –reactive	10	10 hrs	FTND Male – 2.6 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
133	Madhu	25	P_{2+0}	-	Anaemia BOH prev. 2 still births	37 weeks	Baseline -120/min Beat to beat variability -15 Acc – 4 Dec –none	10	7 hrs	LSCS for fetal distress Male 2.7 Kg APGAR (10) at 5 min No meconium

							Movement – 4 Result –reactive			**Cord twice round neck** No NICU adm
134	Ramjanki	23	P_{0+0}	-	IUGR Oligohydrom nios	36 weeks +3 days	Baseline -130/min Beat to beat variability -13 Acc – 3 Dec –none Movement – 3 Result –reactive	6	9 hrs	FTND Male – 2.3Kg APGAR (10) at 5 min No meconium No cord No NICU adm
135	Dr. Kirti	30	P_{0+1}	-	Gestational Diabetes prev. abortion	37 weeks +5 days	Baseline -135/min Beat to beat variability -12 Acc – 3 Dec –none Movement – 3none Result –reactive	8.5	26 hrs	LSCS for fetal distress Female 2.6 Kg APGAR (10) at 5 min No meconium No cord No NICU adm
136	Radha	25	P_{0+0}	-	Anaemia	37 weeks	Baseline -160/min Beat to beat variability -3 Acc – none Dec –none Movement – none **Result – Non reactive**	10	14 hrs	FTND Female – 2.3 Kg APGAR (5) at 5 min No meconium **Cord once around neck** **NICU adm X 2 days**
137	Astha	23	P_{0+1}	-	Rh negprepg prev. abortion	40weeks	Baseline -150/min Beat to beat variability -15 Acc – +nt Dec +nt (variable) Movement – 5 Result –reactive	12	8 hrs	FTND Male – 3.5 Kg APGAR (10) at 5 min No meconium No cord around neck No NICU adm
138	Shobha	26	P_{0+0}	-	Overdue 16 days	42 weeks +2 days	Baseline -160/min Beat to beat variability -5 Acc –nt Dec +nt Movement – none Result –reactive	8	4 hrs	LSCS for acute fetal distress Female 2.75 Kg APGAR (10) at 5 min No meconium No cord round neck **NICU adm X 10 days**
139	Anjana	18	P_{0+0}	-	-	40weeks	Baseline -142/min Beat to beat variability -15 Acc +nt Dec –nt Movement – 4	12.5	24 hrs	FTND Male – 3 Kg APGAR (10) at 5 min No meconium No cord round neck

							Result –reactive			No NICU adm
140	Anu	24	P_{1+0}	1	Decreased fetal movements	38 weeks	Baseline -152/min Beat to beat variability -25 Acc – none Dec –none Movement – none **Result –suspicious**	12	6 hrs	LSCS for decreased fetal movements Female 3.2Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
141	Sukanti	26	P_{0+0}	-	PET	39 weeks +2 days	Baseline -140/min Beat to beat variability -10 Acc – +nt Dec –nt Movement – 4 Result –reactive	11	30 hrs	FTND Female – 2.75 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
142	Malti	20	P_{0+0}	-	Overdue 7 days	41 weeks	Baseline -108/min Beat to beat variability -4 Acc – +nt Dec +nt (ominous) Movement – none **Result – Non-reactive**	8	1.5 hrs	FTND Male – 2.5 Kg APGAR (10) at 5 min No meconium Cord twice round neck No NICU adm
143	Murti	22	P_{1+0}	1	-	37 weeks	Baseline -124/min Beat to beat variability -20 Acc – +nt Dec –none Movement – 3 Result –reactive	11	35 hrs	FTND Male – 2.4 Kg APGAR (10) at 5 min No meconium No cord round neck CTEV & meningomyelocele +nt baby transfer to paeds surgery
144	Abhilasha	25	P_{2+0}	1	-	36 weeks +5 days	Baseline -138/min Beat to beat variability -4 Acc – +nt Dec –nt Movement – 5 Result –reactive	11	15 hrs	FTND Male – 2.8 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
45	Arti	22	P_{0+1}	-	Decreased fetal movements prev. abortion	38 weeks	Baseline -142/min Beat to beat variability -10 Acc – +nt Dec – none Movement – 5	10	5 hrs	LSCS for decreased fetal movements Female 2.65 Kg APGAR (10) at 5 min No meconium

							Result –reactive			No cord round neck No NICU adm
146	Upma	30	P_{1+2}	1	Prev. LSCS Prev. 2 abortion	40 weeks	Baseline -152/min Beat to beat variability -5 Acc – none Dec +nt Movement – 2 **Result –suspicious**	11	9 hrs	LSCS for prev LSCS with fetal distress Female 2. 5 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm
147	Meena	35	P_{0+0}	-	Elderly primi	38 weeks + 5 days	Baseline -160/min Beat to beat variability -22 Acc – none, Dec – +nt Movement – 1 **Result –suspicious**	9	2 hrs	LSCS for fetal distress & LPV , Female 2 Kg APGAR (10) at 5 min No meconium . No cord round neck No NICU adm
148	Harku	28	P_{0+0}	-	-	36 weeks +3 days	Baseline -130/min Beat to beat variability -12 Acc – +nt, Dec –none Movement – 4 Result –reactive	12	15 hrs	FTND Male – 2.6Kg APGAR (10) at 5 min No meconium No cord No NICU adm
149	Santoshi	26	P_{0+0}	-	Loss of movements	37 weeks + 2 days	Baseline -148/min Beat to beat variability -8 Acc – none Dec –none Movement – none **Result –suspicious**	10	4.5 hrs	LSCS decreased fetal movements Female 3Kg APGAR (4) at 5 min No meconium No cord **NICU adm X 2 days**
150	Sanjana	25	P_{1+0}	1	-	38 weeks	Baseline -154/min Beat to beat variability -14 Acc – none Dec – +nt Movement – 1 **Result – suspicious**	16	23 hrs	FTND Male – 2.5 Kg APGAR (10) at 5 min No meconium No cord round neck No NICU adm

Summary

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Since intrapartum events are estimated to account for 20% still births, 20-40% cerebral palsy and 10% cases of severe mental retardation and it has been observed that intrapartum hypoxia is one of the potential factor involved in the development of handicaps and perinantal death. The goal has been to identify fetus at risk by antepartum and intrapartum fetal fetal monitoring.

Admission test is one such non-invasive test by which by a single trace at the time of admission fetal asphyxia already present at the time of admission can be detected, which is not apparent clinically.

To prove the efficacy of this test in predicting fetal jeopardy in utero and its application in improving fetal outcome present study has been done in Maharani Laxmi Bai Medical College, Jhansi from 'September 03 to August 04'. A randomized prospective study was done. Total of 150 patients were included in the study. Among them 99 were from high risk group. These patients were followed upto delivery in the labour room and important events during labour and outcome were noted in a systematic manner.

Later this data was complied and analysed and following conclusions were drawn:

1. There were 106 patients (70.67%) showing reactive NST, 28 patients (18.67%) showing equivocal results of NST and 16 (10.67%) patients showing non-reactive non-stress test.

2. When the results of admission test were evaluated age and parity wise, no definite relation would be drawn.

3. Baseline heart rate when seen in study group showed most of the cases (80%) falling in the range *i.e.* 110-150 beats/minute described by F1GO guidelines, 1987.

4. Distribution of result of admission test among patients of different gestational age groups showed slightly more chances of test being abnormal in postdated patients >40 weeks when compared with other patients.

5. There were 99 patients with one or more risk factors. There was more risk of test being abnormal in high risk pregnancies but it was not significant when individual risk factors were compared same conclusion was drawn.

6. Amniotic fluid index was calculated in all patients by ultrasound using four quadrant method. It was statistically established that in patients with AFI <10 cm there were more cases with abnormal admission test, when compared with cases with AFI >10 cm ($p<0.001$).

7. There were greater number of cases developing fetal distress later in labour in group of patients with abnormal admission test ($p<0.01$, specificity 78% NPV 73.5%. Although it appeared during the study that patients with abnormal NST developed fetal distress earlier but it was not statistically significant.

8. Association of appearance of meconium in patients with abnormal admission test was found to be highly significant (p<.0001 specificity 78.4% NPV 92.45%, Sensitivity – 68%)

9. It was seen that in patients of abnormal NST there were greater cesarean sections done for fetal distress as compared with patients with reactive admission test but on statistical evaluation it was found to be insignificant.

10. When Apgar score of babies at 5 minutes were evaluated it was established that in patients with abnormal admission test greater percentage of babies showed lower apgar scores (sensitivity 66.67% specificity 73.91%, NPV 96.22%,) and more admissions in neonatalogy unit (specificity 76.74%, NPV 93.39% sensitivity 66.67%) (p<.001).

11. Perinatal mortality was seen more in cases showing abnormal admission test. (R=5.05).

To conclude we can say that admission test can be used as an important non-invasive method to diagnose the fetal compromise present at the time of admission in high risk as well as low risk patients in early labour and can decrease fetal morbidity and mortality by timely intervention.